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TWO CASES OF OPERATION FOR TRAUMATIC EPILEPSY; IN ONE OF WHICH THE LATERAL VENTRICLE WAS WIDELY OPENED; IN THE OTHER, AN ABSCESS OF THE BRAIN, CAUSED BY A PORTION OF BONE BEING EMBEDDED IN THE BRAIN FOR FOURTEEN MONTHS, WAS EVACUATED.

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Case I. *Traumatic epilepsy; wide opening of the lateral ventricle; recovery; recurrence of epilepsy.*—R. S., aged nineteen years, from Cadwallader, Fayette County, Pennsylvania, was admitted to the Jefferson Medical College Hospital, April 14, 1901, at the instance of his physician, Dr. Eustan. His father died after an operation for stone in the bladder; his mother and three brothers and sisters are living and well. He had measles and whooping-cough in childhood and diphtheria in March, 1901. In 1892 he was thrown from a horse, striking on the right side of his head over the occipital and posterior portions of the parietal bones. There was a large lacerated wound of the scalp. The bone was fractured and some of it removed. One year later a piece of necrosed bone was discharged. He is said to have been unconscious for nine days, but when he recovered consciousness there was no paralysis either of motion or of sensation. Seven years later, in March, 1899, he had his first epileptic convulsion (*grand mal*). He has had a number of attacks since then. They are preceded by an aura in which his entire head seems to become filled with blood. In addition to these attacks of *grand mal* he has had very many more of *petit mal*. During the attacks he says that his head always turns toward the left. The convulsions, however, do not begin in any one part of the body, but are universal. In the two years since the first attack he has had about fifteen severe ones in addition to a large number of slighter ones. The last attack was three months ago.

The examination of the eyes, by Prof. de Schweinitz, was negative. The urine was clear, of amber color, specific gravity 1024, reaction acid, urea 1.8 per cent. It contained no albumin or sugar. The microscope showed amorphous urates, a few epithelial cells, and a few leucocytes.

In view of the fact that nothing was done at the time of injury excepting to remove a piece of loose bone, and of the relatively recent

beginning of the attacks of epilepsy, after consultation with Prof. Dercum I decided to do an exploratory operation.

Operation, April 17, 1901. I first turned down a large flap of scalp with the periosteum and exposed two openings in the skull, the larger one of which (3 x 1.5 cm.) I had already recognized through the scalp. The smaller one was one-quarter the size of the first. I first removed the bridge of bone between the two openings and enlarged the entire opening by the rongeur forceps until it was 10 cm. long by 3.5 cm. wide, the long axis being antero-posterior. I then divided the dura, which was thin. Immediately below it in some loose connective tissue ran a vessel of considerable size, which from its situation and direction I judged was the posterior branch of the middle meningeal. This was tied with two catgut ligatures and cut. Below the dura and connected with it by some trabeculæ of loose fibres of connective tissue, and at a depth of about 1 cm. below the dura, was a thin, dark-colored sac, apparently the wall of a cyst. On opening this a considerable amount of fluid (probably several ounces) escaped. I doubled a bit of silkworm-gut like a hairpin, and by this means measured the cavity, finding that I could introduce the silkworm-gut for a distance of 10 cm. I then enlarged the opening in the supposed cyst in order to inspect the character of the wall lining it, and, quite to my astonishment, found that I had opened widely the lateral ventricle, the fornix, and choroid plexus, and the cornua in the floor of the ventricle, all being in full view. I was very naturally astonished, because I had not thus far seen anything of the cerebral cortex, much less any white matter under the gray. Though I had often punctured the lateral ventricle, I confess that when I was confronted by a widely opened ventricle I was very much perplexed how to deal with it. I finally decided, as there would be considerable oozing of blood into the ventricle, that it would be wiser to drain for a time, and accordingly I first inserted a small rubber drainage-tube, and alongside of it a gauze wick, with instructions to the house surgeon, Dr. Jiminez, to remove the rubber tube in four or five hours. The gauze I intended to remove the next day, but he was directed, in case of any serious symptoms arising in the interval, to remove all drainage at once and close the wound. I was rather surprised to find that the escape of so large an amount of cerebro-spinal fluid did not produce any symptom whatever. After my clinic was over, though no symptoms had arisen, I decided that it would be wiser to remove the drainage-tube immediately, as I was afraid of the continuous escape of the cerebro-spinal fluid. Accordingly, after the tube had been in place two hours, the house surgeon removed it. The iodoform gauze I removed the next day. As soon as the gauze was removed there was a jet of bloody fluid which spurted to a distance of perhaps 20 or 25 cm.; in all I estimated that between four and six ounces of fluid were lost before I could check it by pressure. My fear was that, as in a case of hydrocephalus, the loss of cerebro-spinal fluid would probably cause convulsions which might possibly even prove fatal. Nothing, however, occurred; even his pulse rate did not change.

On the sixth day after the operation, finding the flap bulging a good deal and the patient complaining of severe headache, I evacuated two or three ounces of cerebro-spinal fluid by means of a pair of forceps slipped under the flap. The fluid this time was almost entirely clear. On the seventh day he was up and out of bed. The wound required no further interference and healed by primary union. His temperature

on the day after operation rose to 102.4°, and the next morning to 103°. It then fluctuated between 101° and 102° until the eleventh day, when it was down to the normal and remained so. After the evacuation of the fluid on the sixth day his headache disappeared. On May 7th, the twentieth day, the severe headache returned, and he was markedly drowsy, without, however, any bulging of the flap to indicate any increased intracranial pressure. At 4 A.M. of May 8th, and again at 11.45 A.M. and 12.10 P.M., he had three severe epileptic attacks, during which the entire body was scarlet. The hands and arms showed marked convulsive movement more than any other part of the body. On May 10th he left the hospital for his home in excellent physical condition. He had had no later epileptic attacks up to May 24th.

REMARKS. In an elaborate paper presented at the International Medical Congress in Berlin, in 1890, but of which only a résumé was read and published, I collected all the cases then recorded in which the lateral ventricle had been opened. Unfortunately, although I handed it in person to the secretary of the surgical section, it was lost, and I had no copy. I have never been able to spare the time to rewrite it. At the time I was greatly surprised to find that that which had usually been considered a fatal accident was, relatively speaking, harmless.

Before the operation upon the present case, I had already opened, in other cases, the ventricles upon both sides and irrigated the brain from side to side, drained the ventricles for a short time, and punctured them to relieve intracranial pressure a number of times, but I had never laid it widely open, almost as widely as one opens a box by removing the lid. Hence, I was in no little perplexity to know just what to do in this case. The result would seem to prove that I decided rightly, viz., to drain for a time and then to close the wound. I was surprised to see how little effect, in fact I might say no effect, was produced by the escape of so large a quantity of the cerebro-spinal fluid. Whether an adult is less susceptible than a child and a relatively normal person than a hydrocephalic, I do not know, but certainly in this case the escape of the fluid was absolutely without result, saving that which was beneficial in relieving the severe headache.

The cause of the very unusual condition which was found in the brain is quite obscure to me. There was absolutely no cortex and no white substance between the posterior half of the lateral ventricle and the membranes. I use the pleural because I suppose that the thin sac, which was 1 cm. inside the dura, was probably the pia. The space between the dura and the pia was occupied by the trabeculæ of connective tissue in the shape of thin threads, in the meshes of which was considerable cerebro-spinal fluid.

Whether the case was one of acquired porencephaly or whether there was simply destruction of the cerebral tissue between the ventricle and the dura at the time of the accident, or by absorption later, I cannot guess. The young man's mental condition when he entered and when

he left the hospital was entirely normal. There was no paralysis, and, moreover, in spite of the extensive and deep destruction of the brain tissue there was no hemianopsia. From the operative point of view his recovery was very gratifying. The brief later history would seem to show that probably there will be no benefit so far as the epilepsy is concerned.

CASE II. *Gunshot wound of the head followed by hemiplegia, traumatic epilepsy and abscess of the brain, due to the embedding of a portion of the bone in the brain for fourteen months.*—H. M. L., aged twenty-one years, of Pittston, Pennsylvania, was admitted to the Jefferson Medical College Hospital, March 24, 1901. His family and personal history have no bearing on the case. He was a private in the Eighth United States Cavalry in Cuba. In February, 1900, while acting as a marker at target practice, he was struck in the head, on the right side of the vertex, by the ball from a Krag-Jorgensen rifle. The ball passed completely through the head, emerging on the other side, and must have passed either directly through or immediately below the superior longitudinal sinus. Possibly the ball passed just above the sinus, destroying the bone, but this seems to be a less likely probability. He remained unconscious for six weeks, at which time his epilepsy began. This was most marked on the left side of the body, including the face, the attacks recurring every three or four weeks. He was not unconscious during the attacks. At the end of March, 1900, an operation was done, a portion of bone being removed. Later, at Santiago, a second operation was done, and a piece of bone inserted to close the opening. In November, 1900, he returned to the United States. His general condition was much improved, and he was able to walk, although the left side of the body was very weak. The epileptic attacks now began to increase in frequency, occurring as often as every six or eight days. They were accompanied with intense pain in the left leg and arm, both of which extremities became markedly weak. In addition to this he suffered from almost constant pain in the back of his head. On January 16, 1901, he was operated on for a third time, the plate of bone being then removed. Since this operation only one epileptic attack has occurred, although he has had threatenings of several. The pain in the back of his head and the paralysis of the left side were not relieved.

On admission his general condition was only fair, his appetite was poor, and the bowels constipated. The left leg and arm and the left side of the face were paralyzed, accompanied by a spastic condition of the parts involved, evidently due to a lesion of the motor area. Prof. Dercum examined him on March 27th, confirmed the observations made, and noted also that there was no Babinski reflex; that the knee-jerk on the right side was slightly exaggerated; that there was no sensory loss. He concurred with me as to the desirability of an exploratory operation. The urine was turbid, amber color, specific gravity 1022, reaction acid, with a slight trace of albumin, no sugar, urea 1.3 per cent. The microscope showed calcium phosphates, amorphous urates, and a few leucocytes; no blood or casts. On admission he was unable to walk, but mentally was fairly clear. From day to day, however, he became more and more somnolent. The temperature, which had risen considerably, had almost immediately fallen to a little below

the normal, and remained there. Moreover, his headache and his epilepsy were increasing in severity. It seemed, therefore, probable that there was a cerebral abscess and that immediate operation should be done. The opening in the top of his skull was 5 x 3 cm., about two-thirds of it being to the right of the middle line, the axis of the opening being almost directly transverse. The superior longitudinal sinus would be a little to the left of its middle and at right angles to its axis. On March 25th Dr. de Schweinitz examined his eyes, with the following results:

O. D., pupil reactions normal, nearly round disk. A spot of white tissue on the nasal side, probably congenital; fundus in good condition. O. S., a similar condition, and disk slightly congested. He has a history of having had diplopia, but at present there is neither paralysis of the ocular muscles nor hemianopsia.

Operation, March 30th. I made a large horseshoe-shaped flap on the right side, the highest point of the curve being just to the left of the middle line. Without much difficulty I dissected away the flap from the dura. As soon as the dura was exposed it bulged very distinctly through the opening. Through the dura a hard mass about the size of the last joint of the finger was felt, about the nature of which I was very uncertain. I then enlarged the opening in the bone about 1 cm., opened the dura parallel with the bone, and turned back the flap, the base of the flap in the dura being toward the middle line, so as not by any accident to open the sinus or the parasinoidal spaces. The hard mass above referred to, which was now found to be about 2.5 cm. in diameter, lay in the brain just at the upper end of the fissure of Rolando. On incising the mass to discover its character the knife impinged on a hard, rough substance which I judged to be possibly a portion of the ball, but more likely a piece of bone. I then was able, partly with my finger and partly with a scoop, to remove the entire mass, finding it to be 4 cm. in depth. On laying it open a fragment of bone, rather less than the size of the last joint of my forefinger, was found in its interior, the hard mass being made up partly of the bone and partly of the hard fibrous tissue in which it was encysted. On inspecting the bone one side of it showed the serrations of the sagittal suture; evidently it had been driven into the brain at the time of the accident, in February, 1900, and had remained embedded in the bone nearly fourteen months. At the bottom of the cavity left by the removal of the bone and its surrounding tissue, I evacuated an abscess which contained about one-half ounce of pus. From the surface of the brain to the bottom of the abscess was 7 cm. Although I feared from the position and depth of this cavity that it communicated with the lateral ventricle, I could not establish the fact, and think that the ventricle most probably escaped. After washing out the cavity with normal salt solution I packed a little iodoform gauze into it, and in closing the wound led the gauze out obliquely under the flap instead of making an opening in the flap directly over the gauze, my object being to avoid, if possible, a fundus cerebri by the oblique drainage.

After the operation the temperature usually fluctuated between 97° and 99°. The packing was removed on the second day, and a small bit of gauze inserted again obliquely for 2.5 cm. under the flap. This was removed on the fourth day. On April 10th, the eleventh day, the temperature suddenly rose to 100°. A pair of forceps was inserted under the flap evacuating a few drops of pus, when the temperature

again fell to between 97° and 98°. On April 14th, the fifteenth day, the temperature again rose to 101.4°. After the evacuation of a few drops of pus the temperature again fell to between 97° and 98°, which seemed to be possibly his usual temperature. He went home on May 8th. Four days after the operation he could move his left leg, and about the same time motion returned in the left arm. His use of these extremities steadily gained, so that on April 20th, the twenty-first day, he was out of bed and could walk quite well, although the left ankle was still weak. When discharged, on May 8th, the wound had been entirely well for a number of days. He occasionally suffered from moderate, but never from severe, headache. His general health was perfect, both physically and mentally, although for several days after the operation he had been almost entirely unconscious.

REMARKS. Perhaps the only remarks necessary in this case are to point out the fact that at three operations by some accident the portion of bone embedded in the brain tissue had not been discovered; that it had produced an abscess over a year after the accident is also noteworthy; and that it was not a bit of the bone plate inserted at one of the operations, but was a portion of the original bone driven into the brain at the time of the accident was proved by the serrations of the sagittal suture. It is entirely too early to predict anything as to his epilepsy.

PRIMARY TUBERCULOSIS OF THE PERICARDIUM.*

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TUBERCULOSIS of the pericardium presents itself (*a*) in the form of scattered miliary tubercles; (*b*) as a tuberculous infiltration, with great thickening of the pericardial layers; or (*c*) as a serous, serofibrinous, purulent, or hemorrhagic pericarditis. In miliary tuberculosis the nodules are found most plentifully near the base of the heart, or on the parts reflected upon the great vessels. The process is, as a rule, merely an incident in a general tuberculosis, and possesses no clinical importance. The tuberculous infiltration is either associated with abundant serofibrinous exudation or with more or less extensive cohesion of the pericardial surfaces, and not infrequently with great thickening of the layers and complete obliteration of the sac. The heart then looks as if ensheathed in a cuirass (Merklen[1]). On cursory, naked-eye examination the appearances are often not in the least characteristic of tuberculosis. Sometimes, if the process is recent, a careful separation of the layers may reveal here and there miliary tubercles or larger cheesy conglomerates; but in old cases of pericardial symphysis the familiar picture of tuberculosis may be so completely obliterated that the most careful

* A paper, somewhat amplified, read before the Pathological Society of Philadelphia, December 27, 1900.

inspection with the unaided eye fails to reveal the true nature of the process. It is only the microscope that under these circumstances can definitely demonstrate the tuberculous character of the pericardial disease. In rare instances, as in one reported by Kast,[2] tuberculosis produces a purulent pericarditis. A very unusual case is also recorded by Eichhorst.[3] The pericardium was the seat of three ulcers that had all the appearances of tuberculous ulcers, such as are seen in the intestine. In many instances the fluid is hemorrhagic. This was so in the remarkable case of Musser,[4] in which the pericardium contained sixty-four ounces (two litres) of blood. In the analogous case of Hirtz[5] the sac was filled with bloody fluid, amounting to nearly three litres (2790 grammes).

From the point of view of etiology, we may divide tuberculosis of the pericardium into the following forms:

1. That which is a part of a general miliary tuberculosis.
2. That associated with general serous membrane tuberculosis (tuberculous polyserositis).
3. That due to extension from neighboring organs, as the lung or pleura, the mediastinal and peribronchial lymph glands, the bones (vertebræ, sternum, ribs), and the myocardium.
4. That developing independently.

The first needs no special consideration, as the pericardial tuberculosis is entirely subordinate to the terminal systemic infection. Pericarditis as a part of general serous membrane tuberculosis is not common. More often the pleura and peritoneum are involved together, the pericardium escaping, but in rare instances all three serous membranes are diseased. The tuberculosis in these cases presents itself either as an acute miliary tuberculosis or as a chronic fibrous process.

The majority of cases of well-marked pericardial tuberculosis are the result of extension from neighboring tuberculous foci—most frequently from the mediastinal and peribronchial lymph glands. Weigert,[6] to whom we owe the demonstration of this fact, showed that the pericardial disease, as a rule, has its origin from lymph glands situated on the anterior layer of the pericardium, in the anterior mediastinum, or from those found at the point of reflection of the pericardium. In the case of Kast,[7] previously mentioned, a gland belonging to the group placed about the aorta and the pulmonary veins, near the posterior surface of the pericardium (the sub-bronchial glands of Baréty[8]), had perforated into the pericardial sac.

Ordinary pulmonary tuberculosis does not often lead to pericarditis. Leudet[9] found it only eight times in 299 autopsies. In the experience of Willigk[10] it was still rarer—eleven times in 1317 autopsies in phthisical cases. Louis[11] found it three times in 112 cases.

In the presence of well-marked tuberculosis of any of the neighboring organs we should always suspect an associated pericarditis, particu-

larly if obliterative, to be tuberculous in origin; but in the absence of well-marked tuberculous disease elsewhere there is frequently nothing to call attention to the possibility that a pericarditis in a given case may be tuberculous. Hence it is probable that cases of tuberculous pericarditis are often recorded as simple forms of pericardial inflammation. Some of these may belong to the fourth group—that of primary tuberculous pericarditis.

In the case which I have the honor to report the appearances at autopsy were not at all suggestive of tuberculosis, yet the microscope revealed lesions typical of that disease. The patient was under my care at the Philadelphia Hospital during the past summer, and on the termination of my service passed into the hands of my successor, Dr. S. Solis-Cohen, with whose kind permission I make this report.

The man was thirty-two years of age, white, of healthy family, an iceman by occupation. He had led a very intemperate life, but had never had any infectious disease, with the exception of occasional colds. Ten days before admission, while on a debauch, he was seized with pains in the shoulders, chest, and back, with cough and headache, and these were the symptoms upon his entrance into the hospital, May 22, 1900.

Apart from some coarse râles over both lungs, posteriorly, nothing abnormal was noted except rapid breathing and feebleness of the heart sounds. It is particularly stated in the ward notes that no murmurs were present. Five days after admission a friction sound was discovered.

When I examined the patient on June 1, 1900, I found him to be a well-nourished, robust, rather irritable man, with a very florid complexion. There was a loud, rasping, superficial, to-and-fro friction sound over an area considerably larger than a dollar in the region of the third and fourth left intercostal spaces within the nipple line (Fig. 1). The friction was not palpable. There was also a pleural effusion on the left side, over which bronchial breathing could be distinctly heard. The intensity of the friction sound varied markedly from day to day. On June 3d it was exceedingly loud and galloping; on the 6th its area of audibility had shrunk to the size of a half-dollar just above the apex (Fig. 2); three days later it could be heard only at the base, in the pulmonary area; on June 15th the friction sound had returned in all its intensity, and could be heard to within one and one-half inches of the suprasternal notch above; to the left to beyond the left nipple line; below as far down as the costal border; and to the right as far as the right parasternal line (Fig. 3). The pulse was rapid and very small, feeble, and compressible. The hands were cold and bluish. The apex-beat could be neither seen nor felt. Percussion showed the cardiac boundaries to extend above to the third rib; on the right, to the midsternal line; on the left, to just outside of the nipple line, and below, to the fifth interspace. Despite the pleural effusion there was no difference between the two sides of the chest on mensuration.

Gradually the friction sound disappeared, but the patient remained weak, and his pulse was feeble and rapid. This feebleness of the pulse was one of the striking features throughout the patient's illness. He often insisted upon getting up out of bed, but always became very short of breath and was forced to lie down again. His disposition was curiously morose and peevish, and his face constantly wore an anxious expression.

From June 27, 1900, onward the friction sound remained in abeyance. There was not then, nor at any other time, any endocardial murmur. The sounds were very feeble at the apex, but quite loud at the base. There was no systolic retraction of the interspaces or of the lateral wall of the chest, nor was the pulsus paradoxus noted. The

FIG. 1.

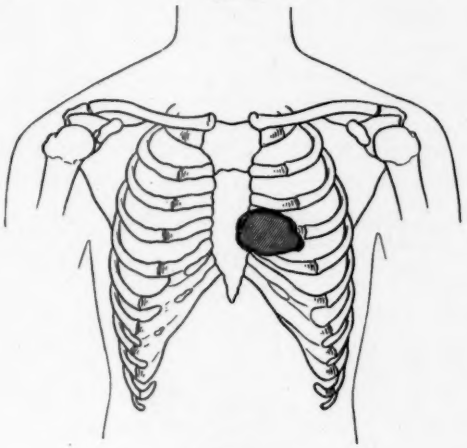
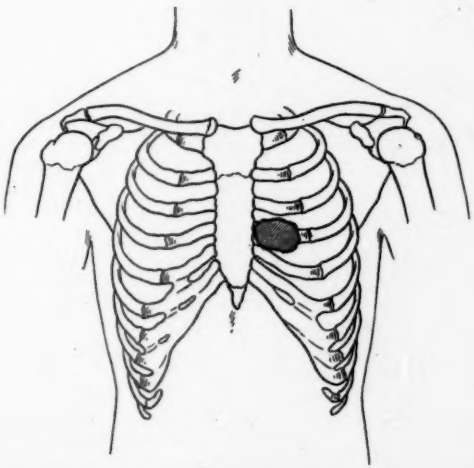


FIG. 2.

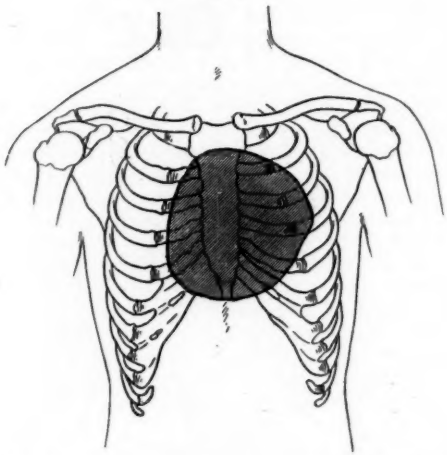


apex-beat could not be detected either by inspection or by palpation. The veins of the neck were not distended. The temperature ranged from normal to 101°, with occasional drops to below normal. The urine contained a trace of albumin, a few hyaline casts, and leucocytes, and had a specific gravity of 1020.

On July 11, 1900, the left pleural cavity was tapped, and a quart of bloody fluid withdrawn. This fluid was examined by Dr. Behrend, of the resident staff. It had a specific gravity of 1012, and was neutral in reaction. Microscopical examination revealed blood-corpuscles and some flat cells, but no bacteria. On the addition of nitric acid the fluid became solid from the precipitation of albumin. Sugar was absent.

After the disappearance of the friction sound the man did not improve, and the diagnosis lay between adherent pericardium and pericardial effusion. The area of cardiac dulness was not changed in shape, although the left border could not be accurately delimited on account of the presence of the pleural effusion, but there was no increase in dulness upward, nor to the right, and Rotch's[12] sign—the appearance of dulness at the sternal end of the fifth right interspace, from obliteration of the resonant cardiohepatic angle—was not present.* It was

FIG. 3.



therefore considered probable that the man had adherent pericardium. The insidious nature of the entire process, and the bloody character of the fluid in the pleural sac led me to suspect that the pericardial disease and the pleural effusion were tuberculous in origin. At my request, Dr. Boston, the bacteriologist of the hospital, was kind enough to inject a guinea-pig with some of the pleural fluid obtained July 11, 1900.

The patient had to be tapped a second time in September, 1900. He failed gradually, ascites developed, the left pleural cavity again filled up, and the tissues in general (including those of the lower limbs, face, scrotum, and penis) became intensely œdematous. Cyanosis was very marked, and became extreme when the patient made any undue exertion (such as sitting up suddenly or attempting to get up out of bed). He also had peculiar syncopal attacks, in one of which I had the oppor-

* No note was made of Ewart's[13] sign. This, also known as the first-rib sign, consists in an alteration in the relation between the first rib and the clavicle, so that the upper edge of the former can be felt as far as its sternal attachment. It is due to a raising of the clavicle at its outer and inner extremities, and is made possible by a relaxation of the ligaments between the clavicle and the first rib.

tunity of observing him. His face suddenly became livid, the pupils dilated widely, and there was a momentary loss of consciousness with twitching of the arms. He automatically raised himself half-way to a sitting posture, the eyes staring. A moment afterward he recovered consciousness and sank back exhausted and sweating profusely. The pupils then contracted, and the face assumed its natural aspect. The whole seizure lasted about half a minute.

The patient had also a severe, dry, rather muffled cough. As a rule the pupils were dilated, and from time to time presented inequality. Light reaction was present, but sluggish. The bowels were regular. The appetite was variable, and there was some epigastric distress after eating. The tongue was usually clean and moist. There were no chills or sweats.

In September the abdomen was tapped for the relief of the ascites, and fifty ounces of a clear, straw-colored fluid were removed. Improvement followed, but it was transitory. On October 11th the patient fell over in the bath-room. He was quickly carried to his bed, but died ten minutes later.

The autopsy was made on the following day. The body was that of a well-developed white male. Post-mortem rigidity was fairly well marked. The face showed some cyanosis; the lips were blue. The abdomen was distended, and the lower limbs were œdematous. On opening the abdominal cavity a large quantity of fluid (1800 c.c.), straw-colored, and having a specific gravity of 1017, was found. A number of old adhesions were present between the visceral and parietal peritoneum. The appendix was coiled upon itself, and the lumen was apparently obliterated. The liver projected 6 cm. below the costal margin in the midclavicular line. The left pleural cavity contained 1850 c.c. of a clear, straw-colored fluid, having a specific gravity of 1015. The lung was collapsed and adherent to the parietal pericardium by a few fibrous strands. The right pleural cavity was obliterated by old adhesions. The spleen was firmly adherent to the diaphragm; it measured 14 x 10 x 4 cm., and weighed 230 grammes. Section showed it to be congested.

The pericardial sac was obliterated, and it was impossible to separate the visceral and parietal layers. The two layers, with the intervening tissue, had attained an enormous thickness, in places as great as 1½ cm.; a large and extremely strong fibrous band, standing out in bold relief from the general pericardial surface, passed from the heart to the diaphragm. There were also some adhesions between the pericardium and the right and left pleura. At their origin the great vessels were embedded in a thick covering, composed of the infiltrated, coherent, pericardial layers. This coating was nearly a centimetre in thickness, and obliterated the natural recesses between the vessels.

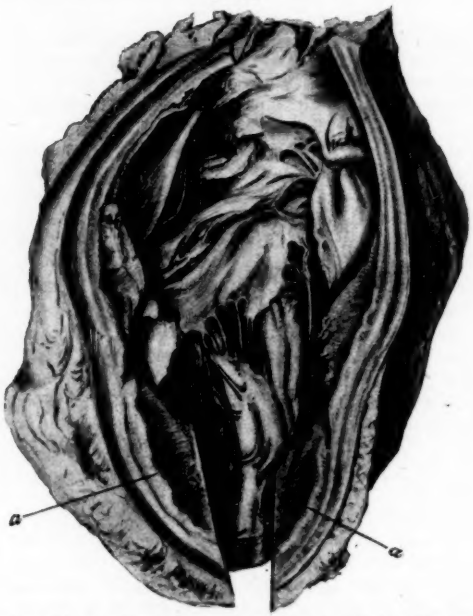
On section it was possible to distinguish three layers in the thickened pericardium—a reddish central layer, and an outer and inner layer, which were paler than the central one and somewhat translucent. Macroscopically there was no evidence of tuberculosis in the pericardium, nor in the pleura or the lungs, except for an old fibroid tubercle, very small in size, in the right apex. One of the bronchial glands was a little enlarged and anthracotic, and on section showed two small, whitish, non-caseous areas.

The heart valves, so far as could be determined without cutting the heart entirely open, were not diseased. The tricuspid orifice easily

admitted four fingers. There was decided hypertrophy of the ventricles, particularly of the right. The left lung was collapsed, and absolutely non-crepitant. The heart and left lung together weighed 840 grammes. The right lung was œdematous, congested, and adherent to the chest wall. It weighed 580 grammes, and contained, as stated above, a fibroid tubercle at the apex.

The liver measured 25 x 16 x 6½ cm., weighed 1450 grammes, and was firmly adherent to the diaphragm. On account of these adhesions its surface was rough; on section the organ was congested, and was the seat of slight fatty infiltration. The gall-bladder was distended with bile; the ducts were patulous.

FIG. 4.



Primary tuberculosis of the pericardium; obliteration of pericardial sac; great thickening of pericardial layers. *a a*. Right ventricle cut open.

The left kidney measured 10½ x 5 x 3 cm., and weighed 140 grammes. The capsule stripped with difficulty and left a rough surface. The organ was firm and congested. The right kidney measured 11 x 5 x 3½ cm., and weighed 160 grammes. In general appearance it resembled the left. The suprarenal glands were normal. The pancreas and intestine presented nothing abnormal, except a little congestion. There were a few peritoneal adhesions.

Pathological Diagnosis. Chronic adhesive pericarditis, hypertrophy of the heart, chronic pleurisy of the right side, passive effusion in the left pleural and peritoneal cavities, atelectasis of the left lung, localized chronic peritonitis.

Microscopical Examination. Pieces from the heart, after they had been for some time in alcohol, were further hardened in alcohol and

embedded in celloidin. Sections were stained with hæmatoxylin and eosin, with Weigert's stain for elastica, and with carbol-fuchsin for tubercle bacilli.

In a section from the right auricle stained with hæmatoxylin and eosin are shown the heart muscle and the two adherent layers of pericardium. Both layers are the seat of an intense inflammation with round-cell infiltration, which is most marked near what corresponds to the surfaces of the pericardial membranes. There is also a decided cell infiltration at the junction of the visceral layer with the myocardium. This is not equally well marked along the entire line of junction, areas of intense inflammation alternating with others where the process is mild. At the base of the epicardium the cells are principally of the small lymphoid type, with deeply staining nuclei; but as the surface is approached large, pale, fusiform or oval cells make their appearance. These vary in size, and their protoplasm can scarcely be distinguished. They are especially numerous along the trabeculæ of the epicardial fat. Mingled with them are a few large round cells with an abundant pale protoplasm and a deeply staining nucleus. The nucleus resembles that of the small lymphoid cell, but the protoplasm is much more abundant than in the latter. As the surface of the epicardium is approached the pale epithelioid cells become more plentiful and more closely packed. As a result of pressure they—*i. e.*, their nuclei—are distorted. They are elongated, and many are rod-shaped. In places the cells have a certain trend, running in long straight or curved lines, sometimes quite parallel. Where this is the case they seem to follow the planes of newly-formed capillaries. At the points of densest cell accumulation giant cells are seen. They are unusually large and numerous, and are surrounded chiefly by epithelioid cells, many of them with irregular, elongated, twisted or sinuous nuclei. Only a few lymphoid cells are seen in the neighborhood of the giant cells. The outlines of the original tubercles cannot be traced, and the entire pericardial layer near the surface is the seat of a more or less continuous accumulation of round, epithelioid, and giant cells, constituting the so-called tuberculous infiltration.

Newly-formed capillaries are fairly abundant in the visceral layer, especially near the surface. They can be traced up to the limits of the tuberculous tissue, but not into it. On the extreme surface, beyond the cellular area, is a thick, homogeneous layer, practically free from cells, and staining yellowish-red with eosin. Here and there some dust-like particles of chromatin are seen. The layer appears as if composed of a dense, compact fibrin. In places it is separated from the cells of the epicardium by areas of caseous necrosis; in others, it borders directly upon the zone of round and epithelioid cells. In clefts of this fibrin a few small and large round cells may be seen; in other clefts there are peculiar round, pinkish bodies, entirely homogeneous, without nuclei, varying greatly in size, and appearing to be masses of hyaline material.

The parietal layer presents the same appearances as the visceral. Aside from a slight increase of nuclei there is but little change in the myocardium.

In the right ventricle the picture is, in a general way, the same as in the right auricle, but the inflammation is more intense in the epicardium, and is particularly well marked at its junction with the muscle. There is a great deal of caseous necrosis, and giant cells are numerous and large, especially in the necrotic area. Very little evidence remains of

a former fibrinous deposit. There is some newly-formed, loose-textured, fibrous tissue, with long, narrow nuclei, the latter running in various directions, but chiefly parallel with the pericardial surface. Toward the myocardium the fibrous tissue is bounded by an accumulation of round, epithelioid, and giant cells, and caseous material. On the other side it merges into the tuberculous tissue of the parietal layer. The myocardium shows an increase of nuclei in the muscle fibres. The nuclei are also in places somewhat larger than normal. Toward the surface there is slight invasion of the myocardium by round cells, which seem to follow the capillaries. There is, however, no sign of any tuberculous process in the heart muscle.

The left ventricle presents nothing materially different from the right. In one of the sections a number of nerve bundles have been included. They are situated in the pericardium, just above the heart muscle. There is nothing abnormal discoverable in them. In their neighborhood is a small, sharply circumscribed cell collection, composed entirely of lymphoid cells. It has the appearance of a minute lymph gland.

Sections were stained for tubercle bacilli, but, probably owing to imperfect preservation, no bacilli could be demonstrated.

In sections stained for elastic tissue by Weigert's method the portion the seat of tuberculosis is found to be entirely devoid of elastic fibres. The wall of the right auricle is very rich in elastica. The fibrils run parallel with and transverse to the muscle fibres. The wall of the right ventricle does not contain nearly so much elastic tissue as that of the right auricle, and the fibres are more distinctly connected with the bloodvessels than is the case in the auricle. The fibres approach the diseased zone very closely, but only a few can be traced into the cell accumulation, in one place running up to but not into a giant cell. The parietal pericardium in both auricle and ventricle contains very few elastic fibres, apart from those in the walls of the bloodvessels. In a section of aorta stained for elastica the tuberculous tissue between the layers of the reflected pericardium is also free from elastic fibres.

The guinea-pig which was injected with some of the pleural fluid weighed about 630 grammes at the time of the injection. It lost weight soon afterward, but only to regain it, and to become heavier than it had been in the beginning. In October it began to fail rather suddenly, and in a short time lost between 250 and 300 grammes. On November 8, 1900, practically four months after the inoculation, it died. At that time the patient had been dead about a month.

At the autopsy on the animal, which was made before the nature of the process in the patient had been cleared up by the microscope, a cheesy focus, containing tubercle bacilli on staining, was found in the lung. The liver was studded with small, whitish nodules, from the size of a pin-head to that of a wheat-grain. They could not be peeled out, and staining with carbol-fuchsin did not reveal the presence of tubercle bacilli in them. The nodules were different from those of ordinary tuberculosis in the guinea-pig, and I was at first inclined to look upon them as an example of psorospermosis. The microscope, however, shows that they are caseous areas, and I have no doubt that the nodules are tuberculous. In the spleen typical tuberculous lesions were found.

A point of interest is the discovery of tubercle bacilli in the pleural fluid by animal inoculation. This fluid had all the characteristics of a

hemorrhagic exudate. As there was no tuberculosis of the pleura demonstrable at autopsy it is most likely that the bacilli had entered the pleural fluid either directly from the pericardium or by way of the blood-stream in the acute stages of the pericarditis. At the second tapping, about two months after the first, the fluid was no longer markedly hemorrhagic, and at autopsy it was distinctly serous.

To all appearances the case of adherent pericardium here reported is one of primary tuberculosis of this membrane. So far as I could determine there was no other site of tuberculosis except in the right lung, and here a small healed tubercle was found, such as is common in the vast majority of bodies that come to autopsy in a large municipal hospital. That this focus was the source of pericardial infection must be regarded as possible. It is well known that the bacteria remain alive for a long time even in old tubercles. I would also not deny that a small tuberculous mediastinal gland might have existed at one time, and might have ruptured into the pericardial sac. There was, however, no evidence of such a source at autopsy. Admitting that either of these possibilities obtained, the pericarditis was, nevertheless, the dominant disease in the patient, in comparison with which everything else sank into insignificance. Hence, it is proper, I take it, to speak of the disease as primary, and it is in this, which we might call the clinical sense, that Osler,[14] Coats,[15] and others also use the term. But it is not impossible that independently of the distant pulmonary focus there may have been a primary implantation of bacilli on the pericardium, without the previous production of tuberculous disease elsewhere. This, of course, would be a true primary tuberculosis—that is, primary in the anatomical sense. Such a condition is, no doubt, exceedingly rare, but there is no inherent reason why it could not occur. Nearly all writers recognize, for example, a primary tuberculosis of the pleura. Why should there not be one of the pericardium? The bacilli in these cases must have a portal of entry, through which they invade the system without leaving any trace at the point of entrance. Such portals may be the tonsils, the gums, the teeth, wounds, and the bronchial and intestinal mucous membranes. The majority of cases of lymphatic and osseous tuberculosis are an illustration of this mode of infection. Baumgarten's theory of latent tuberculosis, which is sometimes invoked to explain such conditions, is not supported by strong evidence.

With the view of determining the frequency and the associations of adherent pericardium, I analyzed the autopsies held at the Philadelphia Hospital during the past three years and nine months. In a total of 778 necropsies there were 60 cases of pericarditis—a percentage of 7.7. This is a much lower proportion than those found by Duchek[16] (15.1 per cent.), Willigk[17] (14.1 per cent.), Chambers[18] (16.2 per cent.), and Taylor[19] (12 5 per cent.); but it is probable that in their statistics milk-spots were counted as pericarditis. Among these 60 cases the pericar-

dial sac was obliterated in 20, or in 33⅓ per cent. Leudet[20] found pericardial adhesions in 58 out of 1003 autopsies, the adhesions being general in 25. This is a percentage of 43. Breitung[21] found complete adhesion in 23 out of 324 cases of pericarditis, or in 7.1 per cent.

In none of the twenty cases at the Philadelphia Hospital was any mention made of the existence of tubercle in the pericardium, but as a large number occurred in cases in which there was well-marked tuberculosis of the lungs we may infer that in many at least the pericarditis was tuberculous. This shows what I have emphasized before—that a careful examination of the pericardium, aided by the microscope, is at times necessary to determine the nature of a pericarditis. Of the twenty cases of adherent pericardium five occurred in connection with a more or less active pulmonary tuberculosis. In a number of the others healed tubercles were found in the lungs. In Harris'[22] table, comprising twenty-five cases of mediastino-pericarditis, I find nine to have been associated with tuberculosis.

The influence of pericardial symphysis upon the size of the heart was a point of active contention during the fertile period in which Corvisart, Laënnec, Stokes, Hope, and others were revelling in the discoveries of the stethoscope. Hope was of the opinion that adherent pericardium always led to compensatory hypertrophy. At present it is generally believed that the obliteration of the pericardial sac need not bring about hypertrophy, although it often does. I think that in dealing with this question we must take into consideration the possible coexistence of valvular disease, which in itself would tend to cause hypertrophy. Of seventeen of the Philadelphia Hospital cases in which the weight of the heart was given there was hypertrophy in seven. In all the pericardium was weighed with the heart, but to allow for this I ruled out as not hypertrophied all cases in which the heart and pericardium together weighed less than 450 grammes. In one of the seven the valves were diseased; in four others there was a generalized atheroma or chronic interstitial nephritis. This leaves two, including the one here reported, in which the hypertrophy could be attributed to the pericardial adhesions. In one case in which no weights are given the heart is spoken of as hypertrophied. Intense atheroma and chronic interstitial nephritis were present, in addition to the pericardial symphysis.

Broadbent[23] has advanced the opinion, which was to some extent also held by Bauer,[24] that the size of the heart is determined by the condition of the organ at the time of the formation of the adhesions. If the heart is dilated at that time it is likely to remain so, and may then undergo hypertrophy. Another point to be considered as influencing the occurrence of hypertrophy is the amount of myocardial involvement produced by the pericarditis in its acute stages. If the heart muscle becomes seriously implicated, as is often the case, hypertrophy

will not be likely to occur. It follows, therefore, that the presence or absence of hypertrophy is dependent upon the myocardium much more than upon the pericardium.

Tuberculous pericarditis may occur at any time of life—in children and in adults, but the primary form seems to be most common in men. Its symptoms are usually those of adherent pericardium, although they may be those of effusive pericarditis. It is the former that is most difficult of diagnosis, and it is the one with which I wish more especially to deal.

The first aim is to make a diagnosis of adherent pericardium. That the condition is tuberculous is more a matter of subsequent reasoning and inference than of actual demonstration by physical signs. In my own case the manifestations were cardiac failure, as shown by dropsy, cyanosis, and intense dyspnœa on exertion; the presence of a friction sound early in the disease; the absence of the signs of effusion and of endocardial murmurs; the imperceptibility of the apex-beat; and the persistently feeble pulse. All these pointed strongly to adherent pericardium, but they are not the only symptoms of the affection. Hayem and Tissier[25] have called attention to the absence of impulse, without any augmentation in the area of præcordial dulness, as an important sign. Morgagni was apparently familiar with this, and noted absence of the impulse in thirty out of forty-five cases. Systolic retraction of the interspaces in the apex region, or of the entire præcordia, is often present, but, being met with under other circumstances, it cannot be considered as either an important or a valuable sign of pericardial symphysis. In certain cases in which there is a marked mediastinitis, with adhesion of the heart to the chest wall in front, the so-called Broadbent's sign[26] may be developed—that is, a marked retraction of some of the ribs on the lateral or posterior aspect of the thorax. As already mentioned, it was not present in my case, probably because there was no adhesion between the heart and the chest wall. The sign is most readily detected when the patient is sitting in a good light, and the movements of the chest are carefully observed from a short distance off—first from the front, and then from the lateral aspect. In rare cases there is a definite systolic shock or concussion, which is felt by the hand as a back-stroke. It follows immediately upon the systolic succussion, and is in proportion to the force of the latter. It may be perceptible only at the apex, or over one or more interspaces; sometimes it is discernible over the entire præcordia, even around the left side to the back. It is attributable to the elastic recoil or rebound of the chest wall at the beginning of diastole, as soon as the systolic dragging force has ceased. Its presence is naturally only to be expected when the pericardium is firmly adherent to the anterior wall of the chest and the heart is acting powerfully. Another symptom is a complete arrest of the respiratory movements in the epigastric triangle. This was never absent in Broadbent's cases of adherent pericardium,

but is, nevertheless, not pathognomonic, as it may occur in other conditions. Adhesion of the heart to the chest wall also prevents the postural change of the apex-beat if the latter is present. When, as is often the case, the lungs are adherent to the outer surface of the pericardium, no change occurs in the area of cardiac dulness when determined in inspiration and expiration (Williams[27]).

The pulsus paradoxus, first described by Kussmaul,[28] is not a characteristic sign of adherent pericardium, and was not present in my case. It consists in a marked diminution in the volume of the pulse during inspiration. When present in pericarditis it is probably due to a compression of the aorta by traction of adhesions during the inspiratory expansion of the chest. Another vascular phenomenon of comparatively little value as a diagnostic sign is diastolic collapse of the veins of the neck—the so-called Friedreich's[29] sign. In one case Broadbent[30] was led to a diagnosis of adherent pericardium by the discovery of the systolic emptying of a vein on the front of the chest to the right of the sternum. The explanation suggested is that the pericardium was adherent to the chest wall and heart, and dragged apart the walls of the internal mammary vein during systole, causing a suction action, so that the blood was drawn into its lumen from the afferent veins during systole. A similar case was met with by Roberts.[31] Letulle[32] considers epistaxis an important symptom of adherent pericardium, an opinion shared by Hayem and Tissier, and by Franck.[33] The latter thinks—somewhat strangely, it seems to me—that left-sided epistaxis is especially important.

There is another symptom that deserves mention, and that is the early appearance of ascites, before the onset of œdema of the lower extremities. This particular phenomenon has attracted a good deal of attention since Pick endeavored to establish as a distinct clinical entity a condition characterized by ascites without cirrhosis of the liver, but associated with adherent pericardium (Pick's[34] pericarditic pseudocirrhosis of the liver). The complex had been previously recognized by Strümpell,[35] Curschmann, and others. It is unnecessary to enter into a consideration of the subject here, but there are sufficient observations now extant to suggest that in cases of ascites of obscure origin it is always wise to suspect the existence of adherent pericardium. With such a suspicion in mind confirmation can be more intelligently looked for.

The disposition of our patient possesses considerable interest. It was, as already stated, unusually morose and even suspicious, and I thought it not unlikely that it might be connected with the pericarditis. Mental disturbances are apparently very common in pericardial disease —much more common, indeed, than in disease of the endocardium.*

* Psychic disorders are not infrequent in aortic regurgitation, but accurate statistics are lacking with regard to the incidence of such disturbances in the different affections of the heart.

There is no single type of psychic disturbance that can be called characteristic. In one case that came under my observation there was wild delirium. Da Costa[36] has noted the occurrence of melancholia as well as delirium in the disease, and in a clinical lecture urged that in every case of mania and delirium the heart should be examined for signs of pericarditis. Fever is usually present; it is irregular and not characteristic; according to Vierordt it may be absent.

The symptoms enumerated above are capable only of pointing to the existence of adherent pericardium, and do not distinguish between the tuberculous and the non-tuberculous forms of that affection. It is a curious fact that in nearly all the patients in which such a tuberculosis as I have described was found the phthisical habitus was entirely absent —a point to which Vierordt has called particular attention. As a rule, also, the family history is negative as regards tuberculosis.

The diagnosis of tuberculosis, that of adherent pericardium having been made, would, therefore, depend upon the exclusion of the ordinary causes of adherent pericardium, such as rheumatism and other acute infections. The presence of a hemorrhagic infusion in the pleural cavity would be of signal value in diagnosis. In rare instances it may be possible to establish the true nature of process by animal inoculation. As an aid in the differential diagnosis I append a table, which is in part based upon that of Weill:[37]

Rheumatic Pericardial Symphysis.	*Tuberculous Pericardial Symphysis.*
Heart always enlarged.	Size of heart usually normal.
Dyspnœa more or less constant.	Dyspnœa slight, brought on by exertion.
Palpitation common.	Palpitation rare.
Impulse strong.	Impulse feeble, and not easily detected.
Heart sounds irregular and strong.	Heart sounds irregular and feeble.
Endocardial murmurs common.	Endocardial murmurs absent.
History of rheumatism.	Occasional presence of pulmonary tuberculosis, or enlargement of the mediastinal or bronchial glands.
Other serous membranes usually healthy.	Other serous membranes may be affected.
Fluid present in serous cavities is serous.	Fluid present in serous cavities may be hemorrhagic.

Duration of the Disease. The duration of the disease is given by Vierordt[38] and Weinberg[39] as from four to eight months. In my own case it could be determined with accuracy, and was just five months.

Treatment. The medical treatment of tuberculosis of the pericardium is at present entirely palliative and expectant. Experience at the autopsy table shows that the tuberculous process itself may become

arrested, death resulting more from embarrassment of the heart by adhesions or from degenerative changes in the myocardium than from the tuberculosis *per se*. The boldness with which surgeons now attack the serous cavities and the viscera, including even the heart, justifies the hope that at no distant day in the future they will find a way to break up pericardial adhesions before these have become firmly organized.

The salient conclusions to be drawn from our study may be stated as follows:

1. Tuberculosis of the pericardium is comparatively common.
2. It may be primary in the clinical, rarely in the pathological sense, or it may be secondary.
3. The primary form is either a hæmatogenic infection or is the result of extension by contiguity from some trivial focus.
4. The most frequent source of infection is a tuberculous mediastinal or bronchial lymph gland.
5. The primary form is usually chronic, and appears as an obliterative pericarditis.
6. In a large percentage of cases there is an associated mediastinitis, with adhesions to pleura, sternum, and ribs.
7. The symptoms are those of adherent pericarditis or mediastino-pericarditis.
8. In every case of obliterative pericarditis of obscure etiology tuberculosis should be suspected, particularly if there are no endocardial murmurs.
9. The diagnosis of tuberculosis of the pericardium can usually be made only by excluding other causes, except in rare instances of successful animal inoculation with fluid obtained by tapping a pleural cavity.
10. Tuberculous pericarditis may not present any characteristic features at autopsy; hence, microscopical examinations should be made in every case of adherent pericardium before tuberculosis is excluded.
11. In rare cases a clinically primary tuberculous pericarditis is acute, the exudate being serofibrinous, hemorrhagic, or purulent.

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THE EXTENSION OF AORTIC ANEURISMS INTO AND BETWEEN THE WALLS OF THE HEART AND DISSECTING ANEURISMS OF THE HEART.[1]

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The relations to the heart of aneurisms in the beginning of the aorta are often quite interesting and complex. Interparietal and intraparietal extension may occur, and many so-called dissecting aneurisms of the heart take their origin in the beginning of the aorta.

Dissecting vascular aneurism is the result of an incomplete rupture of the arterial wall from within, through which the blood by its hæmodynamic pressure separates more or less extensively the various layers of the wall. The separation is a rather rough one, there being no real dissection. The walls of the heart are not disposed to this change, because the force or pressure that causes the separation originates in the walls of the heart during their contraction when the various layers are pressed together (Vestberg).

[1] Read by title before the sixteenth annual meeting of the Association of American Physicians, in Washington, April 30 and May 1 and 2, 1901.

Partial cardiac aneurisms are oftener the result of a uniform dilatation of a weakened area than of rupture and dissection. There are, however, quite a number of places where and methods whereby dissecting cardiac aneurisms may be formed by the separation of layers normally more or less adherent to each other. The aneurism need not originate primarily in the cardiac cavity whose walls largely contain it; it may come from other cavities or from the aorta. The sinus of Valsalva may give origin to dissecting aneurisms which extend into the walls of the heart, because, though properly a part of the heart, they are not affected by the physiological limitations for aneurismal formation seen in other parts of the heart (Vestberg). Aneurisms in the beginning of the aorta may extend into the heart, and on the rupture of the original sac secondary cardiac aneurisms of the dissecting variety may develop. Cardiac aneurisms, apparently dissecting, originating in myocardial abscesses, sometimes are not dissecting aneurisms, strictly speaking, because the larger part, yes, even all the dissecting, may have been done by the suppuration; hence, dissecting cardiac aneurism cannot be separated always from aneurism following myocardial abscess. With the foregoing considerations in mind Arthur Vestberg,[1] in his interesting and complete study of dissecting aneurisms of the heart, defines dissecting cardiac aneurism as a pathological cavity communicating with the heart or with the origin of the aorta, due to a separation by the blood of the layers of the walls of the heart.

Vestberg divides dissecting cardiac aneurism into interparietal, septal, parietal, and valvular, the first being by far the most frequent. Of 60 cases (59 from the literature and 1 original) 47 were interparietal or septal, 5 parietal, and 8 valvular. The interparietal is an interesting form, which occurs in the loose tissue under the epicardium around the root of the aorta. Vestberg, who is the first to give an accurate description of this periaortic space, designates it spatium periaorticum cordis. It is bounded externally by the anterior walls of the auricles and by conus arteriosus dexter; internally or centrally by the aorta, the root of the aorta, and the more or less incomplete conus arteriosus sinister. The roof of the space is formed by the epicardium passing from the posterior surface of the aorta to the anterior surface of the auricles and constituting at the same time the floor of sinus transversus pericardii. The space is divided by the coronary arteries into the auriculoaortic and the conoaortic parts (spatium atrioaorticum and spatium conoaorticum). The points of exit of the right and left coronary arteries are designated by Vestberg as apertura spatii dextra et sinistra. It will be seen at once that the space belongs to the heart and that

[1] On dissekerande hjärtaneurismer, Nordiskt Medicinskt Arkiv, 1897, Ny Följd, vii., Nos. 26 and 30.

aneurisms occupying the space must be regarded as cardiac provided they do not possess independent walls from other sources. Ruptures may occur into the periaortic space from conus arteriosus sinister, the sinuses of Valsalva, the lower part of the aorta, and probably from the coronary arteries. Rupture of the aneurism of the left coronary artery, described by Dr. J. A. Capps[1] in my laboratory, might have resulted in a dissecting aneurism of the heart most likely of the parietal type. The thinnest portion of the internal wall is the fibrous part of the conus arteriosus sinister with its prolongations upward between the aortic valves. Vestberg describes a dissecting aneurism of the periaortic space following rupture due to endocarditis of its internal wall between the base of the anterior mitral valve and the aortic valves. Endocarditis, abscess, sclerosis, and other changes in the beginning of the aorta and the aortic sinuses, traumatic ruptures, and the secondary rupture of aortic aneurisms may lead to dissecting aneurisms in this space. The openings are often linear, especially when traumatic, and round when the result of ulceration. At first there may be no distinct wall, but in chronic cases a membrane may form. From the periaortic space the dissection may extend into the interventricular and interauricular septa, the interparietal aneurism becoming septal. Occasionally it becomes parietal. Secondary ruptures into the pericardium, especially at the orifices of the space—the exits of the coronary arteries —as well as into the right heart may take place.

The following specimens illustrate many of the features in connection with this form of dissecting cardiac aneurism and the relation to the heart of aneurisms in the beginning of the aorta.

Laborer, aged thirty years, syphilitic; death from typhoid fever. Aneurism of anterior aortic sinus with extension into wall of right ventricle; beginning aneurisms of aorta.

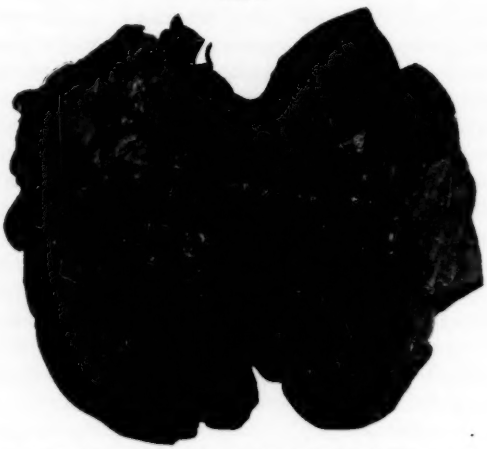
Anatomical Diagnosis (Dr. Crowder). Typhoid ulcer and swelling of Peyer's patches; gumma in epididymis; arterio-sclerosis; aneurism of sinus of Valsalva extending into wall of right ventricle; beginning aortic aneurisms with thrombosis; fibrous perisplenitis and pleuritis; pulmonary œdema; chronic nephritis; œdema of ankles; stricture of urethra; acute cystitis; suppurative prostatitis; catarrhal gastritis.

The sinosal aneurism arises in the sinus behind the anterior aortic valve, the orifice of the right coronary being situated near its bottom; it is 3 cm. in depth and extends into the tissues under the epicardium over the anterior surface of the right ventricle under the apex of the right auricle (Fig. 1). The wall is thin, smooth, extensively calcified, sections showing degenerated fibrous tissue without elastic elements. About 9 cm. above the aortic valves is a brown, circular area, 1 cm. in diameter, which is much thinner than the surrounding tissue and thrown into folds (Figs. 2 and 3), but on outward pressure with the finger a small sacculation is formed. A little higher up is a smaller area of similar appearance and occupied by a thrombus.

[1] The American Journal of the Medical Sciences, September, 1899.

Sections across the depression and including some of the aorta on each side and also the corresponding part of the pulmonary artery, which here is closely apposed and closely adherent to the aorta, show no changes in the pulmonary artery; its elastic network seems perfect.

FIG. 1.



Aneurism of aortic sinus with extension into wall of right ventricle.

At the point of greatest bulging of the aorta the tissue between the aorta and the pulmonary artery is reduced to a thin layer of hyaline fibres; a few bloodvessels are present and also some greatly flattened and compressed nerves; there are but few nuclei in the hyaline tissue and no cell accumulations. On each side of the aortic bulge there is

FIG. 2. FIG. 3.

FIG. 2.—Small sacculation in aorta, showing wrinkling of thinned spot.
FIG. 3.—Small sacculation in aorta, showing thinning of media.

interposed between the two vessels fibrous tissue with areas of fat, nerve bundles, and bloodvessels. Many of the arterioles show a well-marked fibrous, obliterating endarteritis and hyaline adventitia. In some complete occlusion has taken place.

Corresponding to the aneurismal bulging the aortic wall is thrown into folds of varying depth; the endothelium is absent; the wall is thinner by about one-half than on each side of the aneurism; there is hardly any normal muscular tissue in this part of the aorta; the wall consisting of a more or less degenerated fibrous tissue with but few nuclei, often distorted and with a tendency to assume a bluish tinge in hæmatoxylin. This fibrous tissue is in reality the greatly thickened intima. There are no foci of cells in this part, but in the media on each side of the bulging are a few thin-walled vessels filled with blood and surrounded by quite marked cellular infiltrations. The cells have deeply stained round or oval rather small nuclei, and the cell bodies cannot be distinguished. There are no elastic fibres immediately around these perivascular accumulations.

The intimal thickening does not give any elastic fibre stain. There are a few irregular, broken-up bundles and masses of elastic fibres between the thickened intima and adventitia at the bottom of the aneurism. In the sections stained for elastic fibres it is seen clearly how the media is gradually replaced by the thickened intima, the media ending in narrow strands of elastic tissue. The elastic fibres in the adventitia are also largely interrupted over the bulging. The arteries with endarteritis obliterans in the tissue between the pulmonary artery and the aorta show a well-developed network of elastic fibres in the new tissue in the intima.

In the smaller aneurism with the thrombotic plug the conditions outlined above are present in exaggerated degree. There is a marked intimal thickening with degeneration. There is a more sudden interruption of the media, which is completely replaced by the intimal thickening as the outward bulging takes place, on each side of which are masses of broken-up elastic tissue. And the vascularization and round-cell infiltration into the media are more pronounced and associated with similar changes in the adventitia. Owing to the cellular infiltration in the media the elastic elements are broken up extensively. Among the cells in the perivascular infiltrations are seen plasma cells, lymphocytes, and endothelial cells. The thrombus is infiltrated with leucocytes; no bacteria present.

Bacteriological Examination. B. typhosus isolated from the spleen; B. coli from the liver and kidney.

Abstract of Histological Description. The liver contains necrotic foci. There is some increase of the connective tissue in the kidneys and the cells of the convoluted tubules are degenerated; there is some endarteritis. There is myocardial disunion. The spleen contains many phagocytic cells; there are also areas of necrosis. The cell accumulation about the base of the ulcer in the ileum contains endothelial cells with cellular inclusions. There is a chronic interstitial epididymitis with obliteration of the vessels as well as foci of leucocytes and epithelioid cells.

One of the cases of aneurism of the coronary arteries described by Capps[1] presents some features of interest in connection with this subject.

Man, telegrapher, aged thirty-nine years, syphilitic and alcoholic, died from bronchopneumonia developing during hemiplegia and convulsions.

[1] Loc. cit.

Anatomical Diagnosis. Syphilitic leptomeningitis and pachymeningitis; arterio-sclerosis; aneurism of the aorta and of the left coronary artery; chronic aortic endocarditis; fibrous myocarditis; aneurism of interventricular septum; hypertrophy of the heart; chronic nephritis; bronchopneumonia; fibrous pleuritis.

The heart weighs 550 grammes. There is marked thickening of the left ventricular wall. The tricuspid, pulmonary, and ventral valves appear normal. The endocardium is smooth, excepting that the free margin of the aortic valves is rough, thickened, and sclerosed. Just below the junction of the left and anterior semilunar valves is a small bulging in the septum, which admits the tip of the index finger, and is lined with a dense fibrous tissue. Anteriorly the aneurism and the sinus of the posterior pulmonary valve are separated by only a thin partition, which is translucent. The "undefended space" situated to the right of this cavity is unaffected.

Above the left aortic semilunar valve is a bulging of the aortic wall upward and forward into the periaortic tissue. Bordering on this aneurism is the left coronary orifice, which is of normal size. Immediately beyond the opening, however, is a fusiform dilatation of the wall of the coronary artery, large enough to contain a hazel-nut, and extending for the most part posteriorly and to the right. Its walls are streaked with yellowish spots, and in places calcareous scales can be peeled off. Beyond this cavity is a second and smaller dilatation of the wall.

The beginning of the aorta is much thickened, the intima irregularly fissured, and similar changes exist throughout the entire thoracic aorta. The microscopical examination showed principally chronic changes with calcification.

The presence of syphilis in these two cases brings forward the relations of syphilis to aneurism. This is a vexed question. Heller[1] champions the existence of a pure syphilitic aortitis that may cause aneurisms. Peculiar circumscribed depressions, often in groups, especially at the beginning of the aorta, in which the aortic wall is greatly attenuated and wrinkled, with cellular accumulations about the vasa vasorum and destruction and replacement of the elastic and muscular elements of the media, are regarded as the result of a primary syphilitic mesaortitis by Heller and his pupils.[2] In such lesions so-called miliary gummas with giant cells are described by Backhaus and others. Weber has described a gumma as large as a bean in the wall of the pulmonary artery. Backhaus found mesaortitis of this character in seventeen of ninety-nine syphilitics examined at the Kiel Institute. Irregular ulcerations in the aorta with callous margins are also attributed to syphilis. When combined with ordinary arterio-sclerosis the aortic wall may be greatly thickened, uneven, and wrinkled. This is the sclerogummous aortitis of Malmsten, who gives the frequency of syphilitic history in aneurism as 80 per cent. In 82 per cent. of eighty-four cases of general paralysis Straub[3] found the changes described by Heller as syphilitic aortitis.

[1] Verh. deut. path. Gesellsch., 1900, ii., 346–351.
[2] Döhle, Dissertation, Kiel, 1885.
[3] Verh. deut. path. Gesellsch., 1900, ii., 351–363.

Straub also records the presence of giant cells and gummous productions in the media. In some of these cases were aneurisms with peculiar punched-out orifices. Of seventy-one non-paralytics examined during the same period such changes were found in the aorta but seven times, all in syphilitics, however. Ponfick,[1] Ziegler,[2] and Orth[3] doubt the syphilitic character of all instances of this kind of aortitis because similar changes occur in non-syphilitics. Baumgarten[4] also assumes a clinical attitude. Ziegler and Babes[5] hold that ordinary microbes, as streptococci and staphylococci, may produce lesions similar to those ascribed to syphilis. The changes described in the aorta of Case I. correspond well to Heller's syphilitic aortitis, upon the basis of which it is probable that aneurisms in young syphilitics arise. Perhaps the nearest approach at the present time to the truth in regard to syphilis and certain aortic lesions and aneurisms is embodied in the Scotch verdict, " guilty but not proven."

The beginning aneurisms in the aorta in Case I. show in an interesting manner the histogenesis of aneurism, destruction of elastic elements in the media, thickening with new formation of elastic fibres in the intima, and dilatation. It is rare that one has the good fortune thus to trace the starting of aneurism. Rupture of the aneurism of the aortic sinus might have given rise to parietal dissecting aneurism of the right ventricle. This event is illustrated in a striking manner by the following specimen in the collection of the laboratory, which also shows the gross changes regarded as indicating the syphilitic nature of certain aortic lesions and consecutive aneurisms.

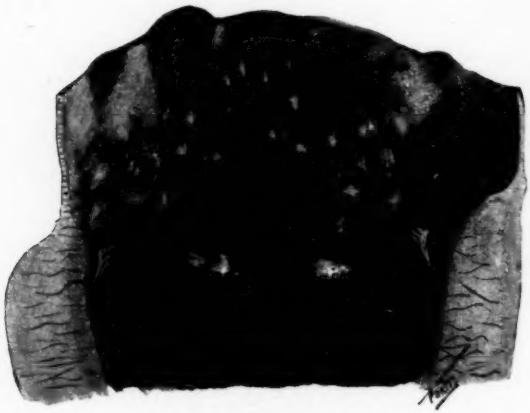
Two sacculated aneurisms of aorta and aneurism of aortic sinus with interparietal and intraparietal extension into heart (Fig. 4). *Dissecting aneurism of periaortic space. Museum specimen; no history.* The heart as a whole is somewhat enlarged, especially the left ventricle. The right heart with its valves is normal. The muscle is of a healthy color. The aortic semilunars are thin and transparent. The beginning of the aorta is dilated somewhat, the intima thick, irregularly nodular and furrowed, and extensively sclerotic. Upon the right and posterior aspects are the openings of three aneurisms. The smallest opening is situated just above the junction of the right posterior and anterior valves; the aneurismal cavity is situated in the periaortic space between the aorta and the right ventricle and auricle; it measures 4 cm. transversely and 7 cm. vertically; the bottom of the cavity rests upon the interventricular septum; at the top the thin roof of the periaortic space is raised quite high. The opening from the aorta into the aneurism is situated about at the centre of its posterior wall. The cavity is empty. There is no trace of the wall of the aorta recognizable in the walls of the aneurism, sections of which show a layer of quite structureless material in the lining, outside of which are a loosely arranged fibrous and fatty tissue with irregular fragments of elastic fibres.

[1] Verh. deut. path. Gesellsch., 1900, ii., 351-363.
[2] Ibid. [3] Ibid. [4] Ibid. [5] Ibid.

The orifice of the second aneurism is a long transverse slit on the posterior surface of the aorta, a little above and to the left of the former. This leads into an irregular cavity composed of two arms—a short one that passes toward the right, and a long one, which passes to the left and downward for at least 4 cm. into the space between the left auricle and the aorta and under the pulmonary artery. The two aneurisms are separated from each other by the descending vena cava, which is closely bound to the aorta at this point. A section of the wall, which is quite thin and covered with thrombi, shows a fibrous structure with a few irregular elastic fibres.

The third aneurism starts in the sinus behind the left posterior valve. The cavity is somewhat irregular and extends into the wall of the left ventricle and along the interventricular groove for 3 cm. At the apex the wall is translucent. The left coronary artery opens at the margin

FIG. 4.



Three sacculated aneurisms of the aorta and aneurism of aortic sinus, with interparietal and intraparietal extension into the heart.

of the aneurism: its descending branch is normal, but the main stem is obliterated for about 2 cm. as it courses over the aneurism. The aortic wall is recognizable throughout in this aneurism.

This interesting specimen illustrates at one and the same time interparietal and intraparietal extension of aortic aneurisms into the heart and the secondary formation of dissecting aneurisms in the periaortic space.

Large parietal aneurism of the right ventricle originating in sinuses of anterior and right posterior aortic valves (Figs. 5 and 6). *Museum specimen; no history.* The heart is much enlarged, the pericardial cavity wholly obliterated, the aorta extensively sclerotic and calcified. The aneurism originates immediately behind the anterior and left posterior valves by an opening 4 cm. in diameter. Viewed from within the aneurism the aortic orifice appears triangular. The aneurism seems to have dissected its way in the wall of the right ventricle, extending

from the aorta between the right auricle and the pulmonary artery to the apex of the ventricle covering the anterior and lateral aspects of the latter. The right auricular appendix is embedded in the wall of the aneurism. At the apex muscular tissue and subepicardial fat are readily recognized upon the external surface of the aneurism, and outside of these is a well-marked layer of fibrous tissue—the adherent pericardium. The lining of the aneurism is almost universally calcified, fissured, roughened, and cracked. On account of the calcification the aneurismal walls do not collapse, but remain stiff, being easily separable into more or less structureless layers in which are also calcareous scales; and there are continuous sheets of calcareous material in the peri-

FIG. 5.

Large parietal aneurism of right ventricle originating in aortic sinuses, anterior view.

cardial adhesions for some distance beyond the bounds of the aneurism. The cavity is empty. There are no definite traces of aortic wall beyond the opening. Microscopical sections from various parts show a structureless wall consisting largely of calcareous material covered externally, especially at the apex, by a layer of fibrous tissue containing a dense layer of elastic tissue like that in the epicardium.

The following specimens are examples of endocarditis of aortic valves as related to dissecting cardiac aneurism. In the first the beginning of the condition is shown, while in the second and third destruction of the internal wall of the periaortic space has been followed by a typical dissecting aneurism in its conoaortic part.

Acute endocarditis of the aortic valves, developing in the course of lobar pneumonia in a man, aged thirty-seven years, destroyed a large part of the left posterior valve, causing an extensive perforation of the segment and extending from the ventricular aspect of the valve to the interventricular septum (Fig. 7). In the septum there is an irregularly oval, ulcerated excavation, covered by detritus. The ulceration has extended upward between the myocardium and the aorta and has reached the lower part of spatium periaorticum (Fig. 7). Microscopical sections through the affected area show about the ulcer an extensive inflammatory infiltration, with numerous colonies of bacteria; the inflammatory changes extend far out into the myocardium and are especially marked in the tissue of the periaortic space.

FIG. 6.

Large parietal aneurism of right ventricle originating in sinuses of aortic valves, posterior view.

The pneumococcus was isolated in pure culture from the vegetations and ulcers.

Acute and chronic endocarditis of aortic valves; dissecting aneurism of the periaortic space (Figs. 8 and 9). The left posterior aortic valve is thick and fibrous, the part near the anterior segment is extensively destroyed, tattered, and shreddy. The right posterior valve shows an oval, irregular ulcer on the ventricular surface. The anterior valve is largely destroyed; it is perforated by an opening, 1 cm. in diameter, from the aortic sinus, and the remaining ventricular surface is covered by granular thrombotic depositions. The triangular space between the anterior and left posterior valve presents an opening coextensive with

the space and reaching up to the upper limit of the attachment of the valves upon the wall of the aorta. This opening leads into a smooth walled, empty cavity, situated between the aorta and the pulmonary conus, and the beginning of the pulmonary artery. This cavity extends to the right and to the left for a short distance from the margins of the opening in the root of the aorta; the prolongation to the left shows a tendency to extend into the interventricular septum. The lower margin of the aneurismal opening, which rests upon the top of the interventricular septum, is quite smooth. The undefended space is not involved in the lesion. The aneurism does not communicate with any other part of the heart than the left ventricle. No trace of aortic tissue is demonstrable in its walls, which in places show a necrotic sur-

FIG. 7. FIG. 8. FIG. 9.

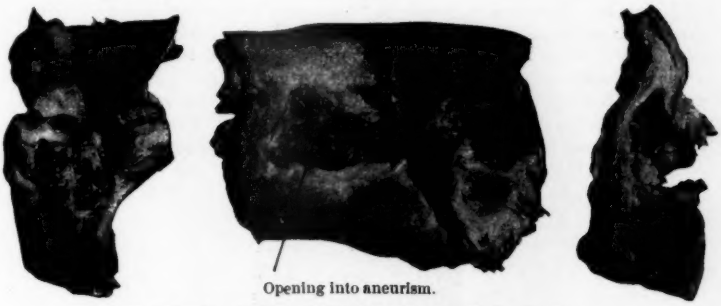


FIG. 7.—Extension of aortic endocarditis to interventricular septum.
FIG. 8.—Acute aortic endocarditis; dissecting aneurism of periaortic space.
FIG. 9.—Aneurism of periaortic space following acute endocarditis.

face with leucocytic and cellular infiltration, especially around micrococcal masses, in the deeper layers. Micrococcal masses are also present in the granular and necrotic layers on the surface.

Acute streptococcus and chronic endocarditis of aortic valves; perforation of internal wall of periaortic space and beginning dissecting aneurism. This occurred in a man, aged thirty-four years, who died from erysipelas and acute streptococcus infection of the aortic and mitral valves during the course of chronic aortic endocarditis. The left posterior and anterior aortic curtains are extensively ulcerated and perforated, and in the triangular space between the two halves is a ragged opening extending into the periaortic space, the walls of which are dissected so as to form a small cavity.

A PERIENDOTHELIOMA OF THE DURA MATER INVOLVING THE CRANIAL NERVES.[1]

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AND

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Report of Dr. Brower.

Mrs. W., aged forty-seven years, was brought to my clinic, October, 1890, by Dr. Annetta S. Dobbins, with the following history: She was admitted to the Woman's and Children's Hospital, May 28, 1882, complaining of a sense of weariness on the slightest exertion, of an intense earache in the left ear, a severe headache, and pain and swelling of the left side of the face. There was a slight trace of albumin in the urine, and a slight aortic regurgitant murmur. She had had three living children and six miscarriages. Under treatment she improved,

Fig. 1.

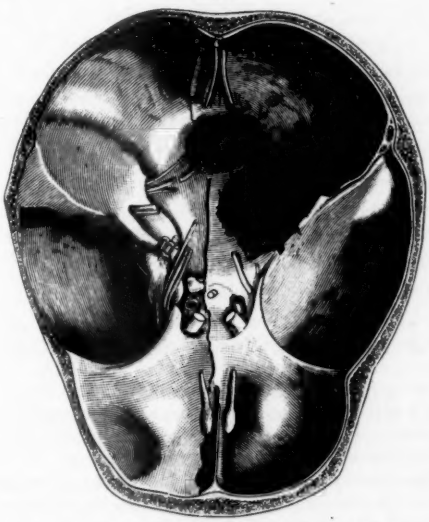
The patient as she appeared at my clinic, December, 1897.

and was discharged June 29, 1882. On January 6, 1883, she returned to the dispensary of the hospital, on account of a distressing and peculiar cough. The following June she again returned to the hospital with a transitory attack of left hemiparesis, the sensory impairment

[1] Presented at the meeting of the Chicago Pathological Society, January 14, 1901, with specimens.

being more marked than the motor, and the cough of the preceding winter more harassing. A month later she had another miscarriage, in which there was an unusual loss of blood. She was again taken to the hospital, and there the facial paralysis was detected, and on examination all the cranial nerves below the fourth except the abducens were found to be involved. On my examination I found paralysis of the motor and sensory branches of the trigeminus on the left side; of the abducens manifested by strabismus, diplopia, and dizziness; of the facial, a complete facial paralysis, the muscles showing the reaction of degeneration; the auditory, with a complete loss of hearing; of the glossopharyngeal, shown by a posterior ageusia; of the pneumogastric and spinal accessory, shown by the cough, disturbed respiration and

FIG. 2.



The lesion diagrammatically as located at this clinic.

heart's action, alterations in the voice, gastralgia, and difficulty in swallowing; of the hypoglossal, by hemiparalysis and atrophy of the tongue. The only symptom that showed progression of the pathological condition was the involvement of the abducens, as compared with the record made in 1883. The lesion began in May, 1882, probably slowly progressed, and just when its maximum was attained, after July, 1883, the record does not show. There was at the time of my examination also atrophy of the left sternocleidomastoideus and of the upper part of the trapezius muscles. Albumin was still present in the urine, and a slight aortic regurgitant murmur, and the arteries showed marked sclerosis. She had had at this time in all twelve miscarriages. The family history was negative; the mother was still living, and the father had died a few months before of cardiac disease.

We made the diagnosis of meningeal tumor, because the symptoms continued to be unilateral until the end. Nuclear lesions are almost invariably bilateral, or soon become so, and, moreover, there was no progression upward or downward. I excluded an amyotrophic lateral sclerosis and progressive bulbar paralysis.

There were at no time in the case any pressure symptoms manifest. Repeated examinations were made of the optic fundus, and nothing abnormal found. Interesting features of the case are, first, its long period of quiescence; beginning with 1890 till the time of her death the case was shown every year at my clinic, and the symptoms showed no progression. The growth of the tumor, also, must have been slow. The next interesting fact is the absence of pressure symptoms, and the third interesting one, and the very important one, is the fact that the pathological study of the tumor excludes syphilis in its pathogenesis, and makes it a benign tumor of the perithelial variety. We supposed it was syphilitic, because of her numerous miscarriages, none of which were the product of violence, and because of the marked arterial degeneration which she had, and because of glandular enlargements, and because of the benefit derived from the use of antisyphilitic remedies. She was probably a syphilitic patient with a non-syphilitic brain tumor, and this fact should make us more cautious in referring the genesis of nervous symptoms to syphilis in syphilitic patients.

The patient died of angina pectoris.

Pathological Report by Dr. Wells.[1]

Autopsy was performed twenty-six hours after death by Dr. S. M. White and myself. The body was that of a slenderly built, poorly nourished woman. It was noticeable that the mouth was drawn to the right and that the left cheek was thinner through its substance than the right. The chief findings in the body were the following:

1. A marked general arterio-sclerosis, seen especially in the larger branches, such as the splenic and coronaries, which were calcified into unyielding, tortuous tubes.
2. Calcification of the aortic valves, without incompetence to the water test or stenosis.
3. Hypertrophy of the left ventricle.
4. Advanced chronic interstitial nephritis.
5. A neoplasm of the dura, extending into the petrous portion of the left temporal bone.
6. Left hemiatrophy of the facial muscles and of the tongue.

Nowhere in the body could lesions characteristic of syphilis be found, although on account of the history of the case and the provisional diagnosis they were looked for most carefully. The extensive arterio-sclerosis and the calcification of the aortic valves could have been syphilitic, but were not necessarily so.

[1] From the Laboratory of Pathology, Rush Medical College.

On opening the cranial cavity nothing abnormal was found over the surface covered by the calvarium. The left inferior posterior fossa, however, was found to be occupied by a mass binding the dura firmly to the skull, less so to the brain. The dura, brain, and tumor mass were removed *in toto* by cutting the growth out of the petrous bone. It was found that this bone had been invaded anteriorly as far as the foramen lacerum medium and along the superior border of the petrous portion of the temporal, and posteriorly to the margin of the foramen magnum, although not into it. The mastoid process had disappeared. This location and extent, it will be noticed, exactly agrees with the provisional location determined from the clinical findings as shown in Fig. 2.

On complete removal the tumor was found to consist of a dense tissue, resembling fibrous tissue in most places, although toward the median line were several softer, reddish, nodular masses. The greatest length of the tumor is antero-posteriorly, this diameter measuring 6.5 cm.; transversely it measures 4 cm., and the same in thickness. More of the growth is external than internal to the dura mater, which seems to bisect it. Its superior surface is in contact with the inferior surface of the left cerebellar hemisphere and the posterior portion of the temporo-sphenoidal lobe, but it is not adherent to the brain nor to the pia mater. It is held attached to the brain by the vessels and nerves entering its substance. On the internal surface it is in contact with the pons and the anterior half of the medulla. It is quite plain why the paralysis involved just the nerves it did—the fifth to the twelfth inclusive. The fourth, rising from its deep nucleus in the floor of the aqueduct of Sylvius, and emerging in front of the pons from the outer side of the crus, would escape any direct pressure from the growth. On the other hand, the fifth, emerging from the lateral aspect of the pons, would be pressed upon at this point, and also as far forward as the Gasserian ganglion. Similarly all the other nerves behind the fifth would be destroyed. Unfortunately, during the removal of the brain the nerves behind the third were torn from the brain at their roots, so that the changes in them cannot be studied.

Connecting the growth to the brain are the anterior and inferior cerebellar arteries and small branches from the left vertebral. Anteriorly the internal carotid enters and courses backward and internally in its substance to emerge near the inner anterior angle. Its walls are much thickened (sclerotic), but not invaded by the tumor tissue, in which it lies in a distinct sheath, nor does the lumen seem lessened by pressure.

The left lobe of the cerebellum is atrophied so that only the posterior half remains, and this remnant is pushed to the right so that its middle line corresponds to the right edge of the medulla, the left tonsillar lobe lying over the lower end of the fourth ventricle. The left temporo-sphenoidal lobe is flattened so that it is about two-thirds the size of the right; this is the result of the lessening in size of the convolutions rather than to a disappearance of them. The medulla is pushed slightly to the right, and the left olive seems smaller than its fellow. The left half of the pons is greatly atrophied, being but 7 to 10 mm. in thickness, while the right is 12 to 18 mm. thick. This atrophy involves the entire left side of the pons. A special study of the changes in the central nervous system will be reported subsequently.

On cross-section the tumor is found to be exceedingly vascular, in some portions almost spongy with bloodvessels. The peripheral por-

tions are less vascular than the central and more fleshy, the centre being rather fibrous. In consistency it is firm, but quite elastic. Many minute granules of calcified material are present near the centre, but absent in the periphery, barely visible to the naked eye as clear, sand-like particles. Except where torn in removal from the bone the growth is distinctly surrounded by a thin, fibrous capsule. The dura loses its identity in the tumor, no traces of it being visible on the cut surface.

Microscopically the different portions of the tumor vary slightly in structure, but are made up in varying portions of bloodvessels, which are always numerous, stroma, and tumor cells. For the most part the vessels are the prominent feature, for in some sections they are so numerous as to resemble an angioma. This is especially true of the peripheral portions of the growth. In the centre the vessels are larger, generally surrounded by considerable connective tissue, and but few of the small vessels are to be seen.

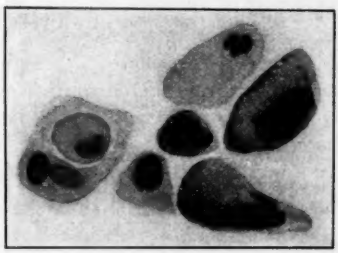
FIG. 3.

Section of tumor, showing its vascular structure and the relation of the tumor cells and vessels.

The larger vessels have walls consisting of an internal layer of endothelium and a series of laminæ of connective tissue surrounding it, with apparently no muscular tissue. They differ from the smaller vessels solely in size and in the amount of connective tissue in their walls. The very smallest capillaries always possess these two layers, an internal lining of endothelium and a layer of connective tissue, which may, however, consist of but little more than a basement membrane supporting the endothelium. No changes were seen in the endothelium, which is identical with that of adult vessels; nowhere is it found proliferating. In proportion to the thickness of their walls the lumen of the smaller vessels is large, so that they resemble the vessels of granulation tissue (Fig. 3).

In the peripheral and most vascular portions the numerous vessels are entirely surrounded by the tumor cells, which fill all the space between them in such a way that it might be said either that the tumor cells form the stroma for the vessels of an angioma or that the vessels form the stroma for the cells In these parts the bulk of the tissue is about equally vessels and cells. Between the cells there is no stroma. The cells are of several types (Fig. 4). For the most part they have a deeply staining spherical nucleus, often eccentrically placed. The protoplasm is quite abundant, and its shape depends entirely upon the surroundings. Sometimes it is symmetrically disposed, and we find a large round or polygonal cell with a relatively small nucleus duplicating a large mononuclear leucocyte or a flat endothelial cell. Again, the protoplasm may be pressed out so that the cell tapers, shaped like a tadpole, with the nucleus at the head. The margin of the cells is always clear, and usually we find a space separating them. Many of the cells are multinuclear, even those that are quite small sometimes having two nuclei. The large multinuclear cells are of various types. The commonest form is about the size of the ordinary endothelial cell when

FIG. 4.



Different types of cells.

viewed on the flat surface, and has from four to eight nuclei, which are usually near one end. Occasionally the nuclei are peripherally arranged, resembling a small, tuberculous giant-cell, but this form is exceptional. The shape varies with the surroundings, but is generally polygonal. When the cells are cut across the nuclei are seen to lie in a mass upon each other, and it is demonstrated that these large cells are usually flat and of the general structure of endothelial cells. Cells are also seen that seem to contain small cells within their protoplasm, resembling phagocytic endothelial cells as seen in lymph glands in the acute infectious diseases. Another type of the large cell has what at first sight appears to be a giant nucleus, but which is in reality a mass of nuclei, superimposed and apparently partly coalesced. A few cells, however, have a single enormous nucleus, staining intensely, very granular or ragged in structure. Probably such large cells as have just been described might be called mononuclear and multinuclear giant cells. Karyomitosis was not observed in a single instance.

As to the arrangement of the component parts of the tumor, the unit seems to be a bloodvessel surrounded by a mass of tumor cells. Whether this vessel is very thinned wall and with a large lumen, as is usually the

case, or is covered by a thick mass of fibrous tissue, does not seem to influence the arrangement. Just outside of its connective tissue walls are always to be found the tumor cells, which are in contact with the connective tissue, but not united to or entangled in it, a distinct line of demarcation being present between the two structures as clearly as between an epithelial structure and its basement membrane. Between one vessel and its nearest neighbor lie only the cells, without any fibrous structure whatever. In other words, as before mentioned, the tumor cells may be considered as stroma holding the bloodvessels in position or the bloodvessels may be considered the stroma of the tumor. The only connective tissue demonstrable by Van Gieson's method is that about the walls of the vessels, or processes which occasionally branch from the larger vessels and the capsule, which run but short distances and seem to connect with vessels.

In the gross specimen calcified spicules were found. It is seen microscopically that these are not simple granules of calcium salts deposited in degenerated tisues, but true bone. None of them is large, and all are surrounded by a fibrous capsule. Within the calcified tissue the bone corpuscles with their branching projections into the canaliculi are plainly seen, most numerous just beneath the investing fibrous membrane. No other evidences of degeneration are found.

Just where to place this tumor as to classification is a matter of much interest. Primarily it must be considered a benign growth, as shown by its clinical history and its gross anatomy. It is evidently a vascular tumor, differing from the simple angiomas in possessing an intervascular matrix of cells of endothelial type. Borrmann[1] has classified the tumors of vascular origin in a way that seems especially clear and satisfactory, as follows:

1. Angioma.

Hæmangioma. Lymphangioma

2. Endothelioma.

Hæm(angio)-endothelioma.
(No capillaries.)

Consists of vessels with a characteristic wall, which are filled with proliferated endothelium. Growth apparently not from new formation of vessels, but only through proliferation of the vessel-endothelium.

Lymph(angio)-endothelioma.
(The common endothelioma of the literature.)

Consisting of cell-cylinders and cell-columns which lie in the lymph spaces. Growth by progression of the proliferating endothelium of the tissue spaces and lymph vessels along the course of the latter.

Capillary-endothelioma.

Consists of new-formed (tumor) capillaries, which arise from proliferated capillary-endothelium. The capillaries become filled finally by growing endothelium. Grows by newly formed capillaries which are protruded as fine tubes or cylinders.

3. Perithelioma and Periendothelioma.

Perithelioma.

Development unknown. Consists chiefly of many vessels with many-layered, superimposed, radially arranged cells, which lie perpendicularly on the outer wall of the vessels.

Peri-endothelioma.

Development unknown. Consists chiefly of many vessels with many-layered, superimposed cells, arranged concentrically about the outer wall of the vessel, with their long axis in the direction of the vessels.

[1] Borrmann. Virchow's Archiv, 1899, Bd. clvii. p. 297.

In the tumor under discussion it is found that the cellular portion always surrounds vessels, and is always the only structure between any two adjacent vessels. These cells do not seem to be in any way related to the endothelium lining the vessels, being always separated from them by a wall of connective tissue. Evidently they form for the vessels of the tumor a perithelial structure, for they lie outside of every vessel and form the tissue separating the vessels. As a perithelioma we recognize under Borrmann's classification a tumor whose cells are of the spindle-cell sarcoma type, arranged more or less radiating from the vessel they surround. Here, however, the cells are not of the ordinary sarcoma type, but are flat cells which, with the exception of being multinuclear in many instances, resemble typical endothelial cells, even to the extent of sometimes acting as phagocytes for other smaller cells. The only cells in the adventitia from which such a tumor can arise are in the lymphatic sheath surrounding the vessel. Such tumors Borrmann calls periendothelioma, which is perhaps as exact and descriptive a name as any for such a growth. He states that in such tumors the cells are arranged parallel to the vessel wall, in contradistinction to the radial arrangement of the perithelioma or angiosarcoma. In this dural tumor no definite arrangement of the cells is seen; they seem to simply fill the spaces between vessels, and may be found cut in every direction in relation to the section of the vessel they surround.

Endothelioma of the dura mater is generally a benign tumor, less often malignant than when arising in the pleura and peritoneum, and still less so than endothelial tumors of the ovary and testicle.[1] It is especially the case that psammomas or sand-tumors are benign, and this tumor macroscopically might be classed as psammoma, although the number of calcific granules was smaller than usually are found in such growths. The calcareous infiltration in psammoma, however, is never an ossification, but simply a deposit in degenerated connective tissue (Sailer). Virchow[2] has recently defined his original use of the term "psammoma," and states vigorously that it does not include the endothelial tumors.

Giant cells have been occasionally recognized in endothelial tumors. Brosch[3] and Glockner[4] have both studied them. The latter describes both the mononuclear and multinuclear types seen in this case, and found them in four of sixteen endotheliomas of the pleura or peritoneum. He considers their origin to be the same as that of the small cells about them—that is, from the endothelium—and due to rapid division of the nucleus without division of the protoplasm. Others, he

[1] For a complete and excellent discussion of the subject of endothelial tumors, see article by Sailer in the Contributions from the Pepper Laboratory, Philadelphia, 1900, p. 416.

[2] Virchow's Archiv, 1900, Bd. clx. p. 32.

[3] Ibid., 1896, Bd. cxliv. p. 289.

[4] Ziegler's Beitrage, 1899, Bd. xxvi. p. 73.

thinks, may form from direct segmentation and fragmentation of nuclei. The large uninuclear giant cells are probably the result of abnormal karyokinesis, and possibly also produced by fusion of a number of single nuclei. Sailer considers that in his case the multinuclear giant cells are apparently aggregations of cells, and many features of my own specimens suggest such an origin. In justice it must be said, however, that identical appearances would be produced by a division of the giant cells into smaller cells, and as a possible confirmation of this view is the much greater frequency of giant cells in the newest parts of the growth. Glockner considers it possible that the giant cells may undergo such a splitting up into small cells. One other source for the giant cells in this case must also be considered. The dura serves not only as a serous membrane, but as a periosteum for the skull, and any tumor arising from periosteum is, of course, especially likely to contain giant cells. In this connection may be recalled the ossified granules previously described. However, giant cells have also been found in endotheliomas not connected with bone—*e. g.*, the pleura, peritoneum, pulmonary vein.

In conclusion, this tumor may be stated to represent a benign growth of the dura mater, derived from the endothelium of the lymphatics of the perithelial layers of the bloodvessels, the periendothelioma of Borrmann. As special structural features may be mentioned the presence of many uninuclear and multinuclear giant cells and spicules of true bone.

GONORRHŒAL MYOSITIS.[1]

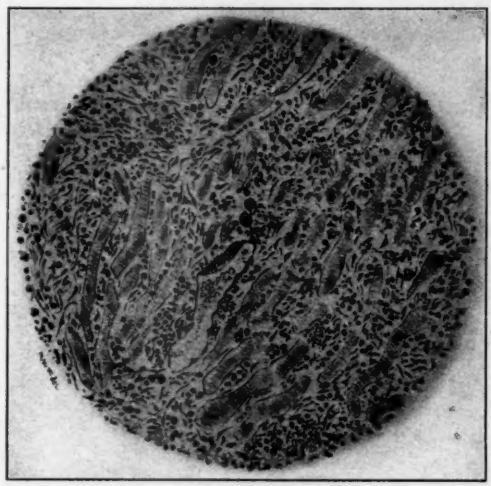
By Martin W. Ware, M.D.,
ATTENDING SURGEON TO GOOD SAMARITAN DISPENSARY, NEW YORK.

The earliest reports of the biological history of the gonococcus accorded it merely the rôle of a local parasite whose habitat in the genito-urinary tract was determined by its predilection for the epithelium here found, though long before the day of the specific gonococcus, clinical experience had taught us of the contemporaneous existence of joint inflammations during the gonorrhœa; yet not till the finding of the gonococcus in the joint secretions by Kammerer, Hartley, and others was the presumed barrier to its growth in tissues other than epithelial broken down. From that time on we have had overwhelming evidence of its ravages, based on its occurrence, in remote parts of the human organism—the heart, nerves (Leyden), and bones (Ullman), and only to be explained by metastasis; but of very recent date are the clinical observations pointing to its affection of muscles. Such a case came under my care.

[1] Read before the Genito-urinary Section of the New York Academy of Medicine, April 17, 1901.

A male, aged thirty-five years, otherwise enjoying good health, contracted gonorrhœa two months ago. In the fourth week of its existence he had a chill and fever with pain in the left knee-joint, preventing him from walking, for which he obtained hospital relief. After three weeks of rest he was discharged (no evidence of joint trouble being present). He consulted me for a severe pain about the shoulder-joint. This region showed fulness, no redness, active abduction was impossible, passively it was attended with instant spasm of the abductor group. While ascertaining this an exquisitely tender area was elicited in the group of muscles which make up the posterior axillary fold. By palpation an induration the size of a walnut was felt posteriorly in the muscles, the overlying skin being movable. From the axillary surface nothing could be felt. The daily elevation of temperature was about

FIG. 1.



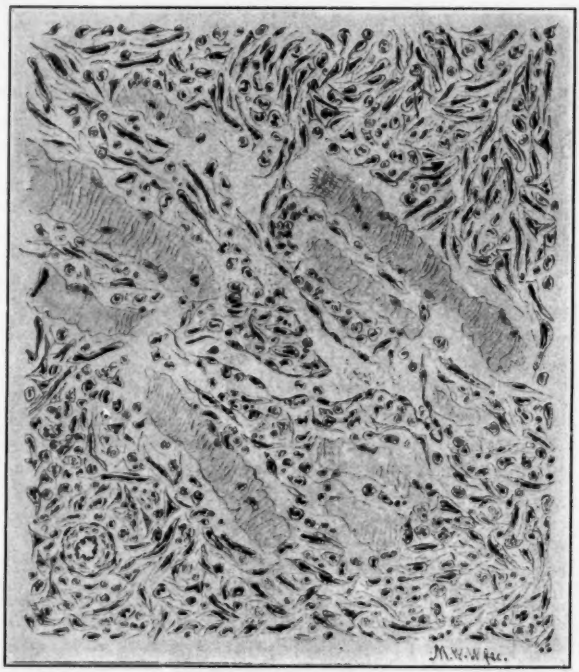
Section of the muscle (3 m.) showing the diffuse extent of the connective tissue proliferation. Ocular 4, objective A A (Zeiss).

100° F. At this stage of the examination the aforementioned history of infection was given, and although the patient subjectively complained of nothing referable to the urethra, yet the glass and Jadassohn tests showed an active posterior urethritis with shreds containing gonococci. Close questioning brought forth an admission of hourly diurnal and three or four nocturnal micturitions. In a few days under expectant treatment (iodine locally) the mass not only grew, but the pain became so intense that I decided to incise in the expectation of giving relief to some pus under tension. Under local anæsthesia I reached the muscles and found them sodden, of a grayish color, and further in depth very friable. No pus was encountered, but a free oozing of turbid serum. Some of the latter material was utilized for bacterial inoculations and cover-glass preparations made. A piece of the muscle was excised.

The wound was left open to be drained, and as much as six weeks elapsed before its final closure. Though relief from pain followed the incision, subsequently the induration extended, though not in degree involving the whole of the muscle, the latissimus dorsi, to its insertion. When the wound finally closed the area of induration retracted very slowly, and in proportion abduction became more and more possible.

Pathology. The only culture media available at the time were sugar agar and sugar bouillon. Two tubes of each were inoculated and remained sterile in the thermostat at 38° C. The cover-glass smears,

FIG. 2.

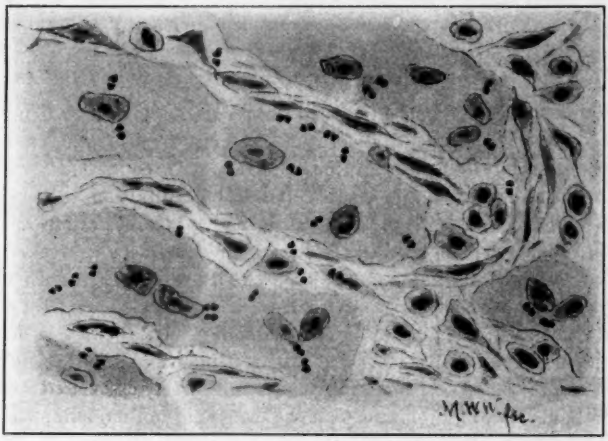


Section of the muscle (3 m.) showing the interstitial connective tissue proliferation and cloudy swelling. Ocular 4, objective D D (Zeiss).

of which three were made, showed diplococci (Fig. 4) in but two, and no other micro-organisms. The diplococci found were typical gonococci in arrangement and shape, and furthermore were decolorized according to the Gram stain. They were exceedingly sparse. Their scant number, together with the unsuitable media, may account for the failure of the culture, but, on the other hand, the exclusion of any other pus organism is valuable evidence of a negative kind in favor of the existence of gonococci. The section of the muscle (3 m.) embedded in celloidin shows an exquisite picture of interstitial inflammation (Figs. 1

and 2). The muscle fibres in part are the seat of cloudy swelling, yet where they are intact the striations are clearly visible. There is some

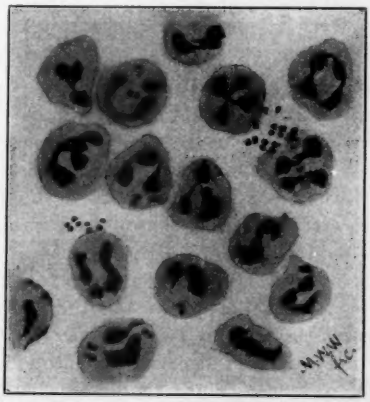
FIG. 3.



Section of the muscle stained to show the gonococci. Ocular 4, objective 1/6 (Zeiss).

shrinkage from the perimysium, and the nuclei in places tend to multiply, which may be a sign of regeneration. The connective tissue

FIG. 4.



Secretion from the muscle. Gram stain, showing the gonococci.

proliferation is so great as to largely increase the interval between the individual muscle fibres and also to compress them. Sections stained

for gonococci (Fig. 3) show nothing of the muscle structure, but a liberal scattering of diplococci grouped about the nuclei and in the interstices of the muscle fibres. The staining was affected by immersing the specimens for thirty-six hours in a carbolic fuchsin solution in the thermostat. This, on the authority of v. Kahlden, was considered the best procedure, since a prolonged immersion of celloidin specimens in a solution of anilin gentian violet water causes them to be digested.

There are recorded in literature three cases of gonorrhœal myositis based solely on clinical observation. Rona[1] reports two cases. A merchant, aged twenty-seven years, during the course of a chronic urethritis, had pain in the thigh followed by induration the size of a silver dollar. The overlying skin was normal. The induration was very tender, appeared embedded in the fascia, and lasted many weeks. The second case was that of a male, aged twenty-seven years, with fourth attack of gonorrhœa. In the second week of its existence pain at the centre of the left thigh set in, but without fever, yet rest in bed was necessary for two weeks. At the site of pain there existed an infiltration the size of a saucer and very tender, intimately associated with the fascia lata, but in no relation with the overlying normal skin.

Eichhorst[2] reports the case of a male, aged fifty-six years, who had a first attack of gonorrhœa. About the fourth week of its existence he complained of severe pain on the outer aspect of the thigh, and a few days later palpable signs of an induration 8 cm. long and 4 cm. wide set in. The temperature fluctuated between 35° and 38° C. Pains, very severe, lasted a week and then abated, but tenderness to touch persisted. The overlying skin was always normal. The infiltration diminished in extent, but gained in hardness. Lead-water applications were of no avail. Two weeks later the right wrist-joint reddened and became tender and infiltrated. These constitute the only authentic clinically recorded cases in literature, though a case of myositis, the basis of an interesting paper contributed by Treves[3] to the London Clinical Society as myositis of questionable origin in a male afflicted with gonorrhœa seven weeks prior, appears at least to belong here clinically, for such was the opinion of two of the speakers on this occasion. It is likewise probable that a large number of cases of myositis reported in females, classed as pyæmic following the puerperium, rest on a basis of gonorrhœal infection, for such is the contention of Koenig for the postpuerperal arthritis in large number. Eichhorst was not willing, owing to the paucity of observations and the want of pathological proof, to set up a clinical picture, yet this appellation of "sclerosing

[1] Rona, S. Ergaenzungsheft 3, Archiv f. Dermatologie und Syphilis, 1892, S. 250.
[2] Eichhorst, H. Deutsch medicinische Wochenschrift, 1899, p. 685.
[3] Treves, F. Transactions of the Pathological Society, London, 1880–1881, vol. xxxii. p. 273.

gonorrhœal myositis" finds justification in the pathological findings for the first time here pictured. The sclerotic process so peculiar to the gonorrhœal process in joints, urethra, and epididymis again manifests itself in the muscles. Bearing on this point is the subsequent course of the new connective tissue formation. In greater part it seems after a variable but usually long period of time to resolve. That the reverse may obtain is gathered from a report by Batut.[1] The latter narrates a case of myositis ossificans of the brachialis anticus in a patient afflicted with gonorrhœa complicated with arthritis. This myositis had its inception in an induration of the entire brachialis anticus. This subsequently became so dense as to intercept the X-rays, and was therefore interpreted as being of bony formation. It was reduced to the size of a nut in four months.

It was in the expectation of finding pus, inferred from the exquisite localized tenderness and the rise of temperature, that I incised. As already stated, merely a profuse turbid secretion escaped which showed leucocytes exclusively and a very few gonococci. The subsequent protracted course of the inflammation, its spread and eventual retraction point clearly to the futility of the incision in stopping the proliferation, though pain abated instantly. From these half-dozen instances cited, together with the pathological findings in this single case of mine, it is safe to predict that notwithstanding the tendency to resolution some stiffening will eventuate from the residue of connective tissue. Pus formation has not as yet been encountered, and if so it will in all likelihood be traceable to mixed infections.

This location of the gonococcus is explained either as a metastasis in the muscle or as an extension of inflammation from adjoining joints or bones. The latter seems more likely, as in most cases the adjacent joints were affected. That bones can be the seat of a gonorrhœal osteomyelitis, the recent experience of Ullman bears testimony. As in the allied affections muscular rheumatism and myositis ossificans we have found an intimate interdependence of muscle inflammation and the oft associated or preliminary joint symptoms, it is not unlikely, reasoning by analogy, that the primary seat of the metastasis is in the joint or bone, and thence follows by way of the lymphatics the planes of the tissues, preferably the tendons.

The treatment will be largely expectant. For the relief of the pain dry heat, hot salt, or sand bags, as in joint disease, will be found to assuage the pain, aided by local anodynes, guaiacol, gaultheria, and menthol. When the acuity of the process has abated massage is in place. If the intensity of the pain be excessive I believe that an incision will accomplish depletion and relieve tension.

[1] Batut. Journal des Maladies Cutanees et Syphilitiques, 1900, p. 273.

DOUBLE URETER OF THE RIGHT KIDNEY.[1]

ONE URETER ENDING BLINDLY; ACUTE SYMPTOMS; OPERATION; DEATH; AUTOPSY.

By Charles L. Scudder, M.D.,
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Anomalous conditions of the ureter are infrequent in viable children. A recognition of such anomalies is difficult. A record of the clinical signs in these cases is, therefore, important. The case here reported is of peculiar interest because it is only the ninth recorded instance of the blind ending of a supernumerary ureter, and it was associated with acute abdominal symptoms.

The case under consideration was that of a child, twenty months old, who was well nourished and had been in good health up to this present illness. During the night of June 25th, without previous warning, the child was restless and vomited.

June 26th. The following day she complained of stomachache and appeared dull and feverish. The bowels moved naturally after taking castor oil.

27th. The child was very ill and presented a pinched and distressed expression. The pulse and respiration were both accelerated, and the temperature was 100° F.

At this time, according to the statement of the attending physician, a sausage-shaped tumor was palpable in the right side of the abdomen. This tumor was soft, elastic, movable, and slightly tender. There was little or no rigidity of the abdominal muscles. A large enema of warm water was returned clear, leaving the tumor unaltered in shape. A few hours later a second evacuating enema was returned clear.

28th. The child was evidently not improving. A diagnosis had not been definitely made, although it was thought probable by the physician that there was present an intestinal obstruction, but a third enema caused a normal fecal dejection. The enemata which came away and the fecal dejection were free from both blood and mucus.

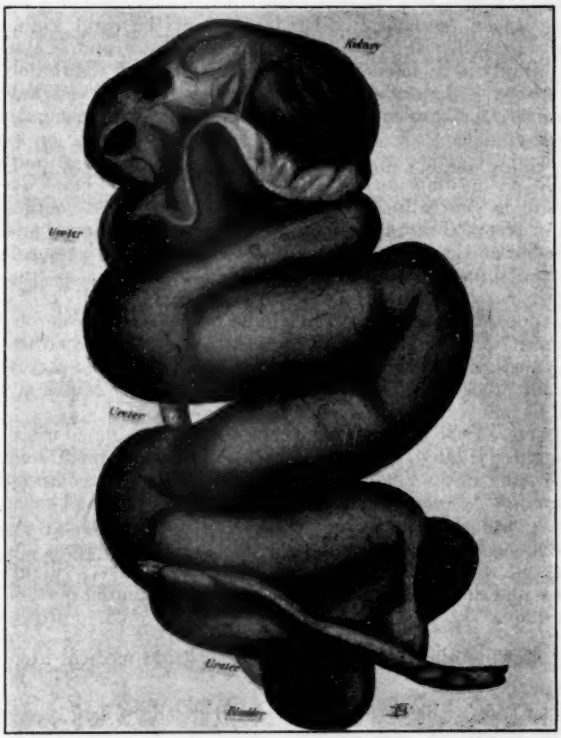
Upon this third day of the illness I saw the child at the Massachusetts General Hospital in the regular rotation of emergency cases. The child was fairly well developed, but with pale, sunken cheeks and dark circles under the eyes. The tongue was moist but slightly coated. The heart and lungs were normal. She was restless and crying out as if in pain. She appeared septic.

The abdomen was uniformly distended, almost everywhere tympanitic and tender. The exception to the tympany was in the right iliac region, where there was great sensitiveness and slight dulness. The muscular rigidity corresponded to this dull area. There was an indefinite soft and elastic oval mass felt, extending from the middle of the iliac crest nearly to the symphysis pubis.

[1] Read at the meeting of the American Association of Genito-urinary Surgeons, Washington, D. C., May 3, 1901.

A suds and glycerin enema resulted in a small fecal dejection. One hour and a half later a high oil enema resulted in another small fecal dejection containing a little mucus. Two observations of the temperature showed it to be fairly constant at 103° F. The pulse was about 140.

A laparotomy was done in the hope of relieving the condition of the child. There was no free fluid in the abdominal cavity. The bowel



The right kidney is seen at the top of the plate split open along its convex surface. The interior of the kidney is shown. The large intestine-like tube (the supernumerary ureter) is seen starting from the right pole of the kidney and ending in a blind pouch outside the bladder. The other ureter is seen starting from the left pole of the kidney and lying behind the greatly dilated ureter.

and peritoneum were normal in appearance. There were no intestinal or omental adhesions. The appendix was normal.

The tumor, palpated before operating, was found to be retroperitoneal, and to consist of a large fluctuating swelling extending from the kidney on the right down in curves across the lumbar region and behind the bladder. The left kidney felt normal. The child's condition preclud-

ing further operation, the abdominal incision was closed. The child recovered from the anæsthetic and died the next morning.[1]

Report of the *autopsy*, No. 118, by J. H. Wright, M.D., Director of the Pathological Laboratory of the Hospital.

Posteriorly beneath the peritoneum and in front of the aorta, extending across the abdominal cavity in the region of the kidneys and lengthwise from the inferior border of the liver to the floor of the pelvis, is a contorted intestine-like resilient mass. Upon dissection this was found to represent an enormously dilated ureter of the right kidney, filled with a thin, yellowish fluid containing leucocytes and bacteria, and ending blindly as a closed sac about the diameter of the thumb, in the neighborhood of the orifice of the urethra, the internal meatus. It has an extremely tortuous course, running back and forth, generally transversely to the long axis of the body and forming four or five loops closely bound together by connective tissue. At its widest portion it is about 11 cm. in diameter. Its length is probably 45 cm. Its lining membrane is generally smooth and white. Above it merges into a dilated pelvis of the right kidney, which has another ureter and pelvis. The portion of the kidney to which the dilated ureter belongs is a portion of the superior pole about 2½ to 3 cm. long. This portion of the kidney tissue shows opaque grayish streaks and is generally of a grayish, fibrous appearance, with little resemblance to kidney tissue on section.

This part of the kidney is about 10 mm. thick. The entire kidney is large, measuring 9 cm. in length. The part of the kidney corresponding to the other renal pelvis shows a slight atrophy of the pyramids. The pelvis and calices are considerably dilated.

The ureter of this portion runs posteriorly to the dilated ureter and is itself slightly dilated, measuring about 8 mm. in circumference, and ends normally in the bladder. The left kidney is not remarkable.

Anatomical Diagnosis. Duplication of right ureter and renal pelvis. Agenesis of the ureteral orifice of one ureter and extensive dilatation, elongation, and tortuosity of the same, with a hydronephrosis and pyelonephritis of the corresponding portion of the kidney. Slight hydronephrosis and dilatation of the ureter of the remaining portion of this same kidney.

The following eight cases are those recorded in medical literature of this anomaly:

Liehler[2] (*Med. Korrespondenzblatt des Wurtemberg ärzll vereins*, Band iv., 1835, p. 23). A girl, three months old, who screamed loudly at every micturition. A tumor presented in the vaginal outlet which had the appearance of a tense bladder. This tumor was replaced, and upon reappearing three days later ruptured. The child died, and the autopsy disclosed a pouch-like blind ending of a supernumerary ureter within the bladder, dilatation of the supernumerary ureter, a partial hydronephrosis of the corresponding portion of the kidney.

[1] See Massachusetts General Hospital Reports, Surgical Records, vol. xix., p. 32.

[2] Carl Schwarz. Ueber abnorme Ausmündungen der Ureteren und deren chirurgische Behandlung. Beiträge zur klinischen Chirurgie. Tübingen, 15, 1896, St. 159.

Lilienfeld (*Dissert.*, Marburg). He describes a preparation from a man, aged sixty-five years. A pouch-like blind ending of a supernumerary ureter within the bladder.

Osterloh (*Jahrbücher der Gesellschaft für Natur und Heilkunde in Dresden*, 1872–3). In the case of a new-born child there was found a left double ureter. The right ureter was normal. The extra ureter ended in a blind sac behind the bladder. The upper half of the kidney was hydronephrotic.

Heller describes a case in a man, aged seventy-nine years, of great hydronephrosis of half the kidney with a double ureter, the blind end of which terminated in the bladder wall.

Boström (*Beiträge zur path. Anatomie der Neuren*, Freiberg, 1884) describes the case of a five-and-a-half-months'-old child who had a double total division of the ureters. One ureter ended blindly within the bladder, compressing the urethra and the other ureteral orifice of that side.

Geerdts (*Dissertat.*, Kiel, 1887) reports the case of a female child, aged three weeks, who presented a tumor, about the size of the end of the little finger, protruding through the urethra. The tumor could be reduced into the bladder. The diagnosis was inversion and prolapse of a part of the posterior bladder wall through the urethra. At autopsy a pouch-like blind ending of a supernumerary ureter within the bladder and urethra was discovered.

Orthmann (*Centralblatt für Gynäkologie*, 1893, No. 7) reports the case of a girl, aged twenty-seven years, in whom the supernumerary ureter ended in a blind sac projecting as a cyst into the vagina.

Stoltz (*Gazette médicale de Strassburg*) describes a case in which the right kidney had two ureters. There was no left kidney. One of the ureters of the right kidney ended in a cystic tumor, the other ended blindly near the bladder.

BRONCHIAL CONCRETIONS.

WITH THE REPORT OF AN ORIGINAL CASE.

By L. W. Atlee, M.D.,
OF PHILADELPHIA.

R. D., aged twenty-nine years, native of Ireland, a fireman in the United States Navy. He is a man of medium height, and very powerfully built and muscled. He was admitted at the Naval Hospital in Philadelphia from one of the ships at the navy yard, the "hospital ticket" giving the diagnosis of his disease as being "bronchitis." Physical examination of the thoracic organs was almost negative—almost, because on the anterior part of the left chest at the second interspace, on the parasternal line, there could be heard some dry,

small, harsh râles. The temperature on admission was 101°, pulse 84, respirations 24. The tongue was slightly coated, and there was some anorexia. The subjective symptoms were, in regard to the lungs, sharp pain, increased by cough, referred to the aforementioned spot, and frequent teasing cough. There was occasionally some thin, slightly yellowish sputum brought up. During the succeeding three days his condition showed no change, excepting that the sputum became freer and slightly purulent. This state continued until the eighth day, when his temperature ran up to 104°, and the respirations and pulse became more rapid. The pain was now much more severe at the spot on the left side. We thought we could detect subcrepitant râles and bronchial breathing, and there was dulness on percussion over the place, measuring some three inches in diameter. On the ninth day the temperature dropped to 101°, and the sputum, which had been tenacious and blood-streaked, became thin and yellowish-green. By the twelfth day the temperature began to show a septic (hectic) character, and the sputum was very profuse and intensely fetid. The stained specimens of the sputum showed no tubercle bacilli, though naturally in such a case they were carefully and anxiously looked for. The general health by this time showed the effects of the disease, as the man, from being robust and ruddy, became sallow, and lost much flesh. The profuse fetid expectoration and hectic continued without change until the fourteenth day. On the morning of this date, during a violent spell of coughing, he felt a solid, sharp body come up with the sputum, which he spit out into his spit-cup. He was astonished at the loud sound it made on striking the bottom of the cup, and called the nurse's attention to it. The cup was brought to me, and the concretion removed. It was about three-quarters of an inch long, and conical in shape, the largest diameter being about one-quarter of an inch. That it looked like a piece of necrosed bone will best describe it, and it had very sharp, rough, and jagged edges. A rough chemical analysis gave the reactions for lime and phosphorus. The man's recovery after getting rid of this concretion was almost immediate, and he left the hospital ten days after its discharge, seeming in no way any the worse for his experience.

It is to be regretted in this case that a more extensive examination of the concretion was not made. The history of this man's condition previous to his admission was entirely negative; he had always enjoyed robust health, and knew nothing as to his having, perchance, at any time got any foreign body in his lung.

The most complete work on this subject was done by L. Leroy, in his *Thèse pour le docterat en Medicine*, Paris, 1868; *Les Concretions Bronchiques*. He eliminates foreign bodies from without, but includes all those which have been found in the respiratory organs under three divisions—those found in the bronchial tubes, in the glands near the bronchial tubes and ulcerating through their walls into their cavity, and those found in the parenchyma of the lung.

Historically the references made to this subject by the ancient authors are very indefinite. Morgagni analyzes the cases published prior to him of concretions found in the lungs or thrown off in the

expectoration. He mentions that Areteus, Galen, Alexander of Tralli, Paulus Aegineta, have observed concretions brought up in coughing; Fabricius cites an example of a consumptive who threw off a great number; Boerhaave says that Vaillant expectorated 400 very small ones; Benevenius saw a stone expectorated that was nearly as large as a filbert; and Cartulus found in the interior of a lung a stone as large as a hen's egg; Liénteaud and Portal bring together a few short observations from the authors who preceded them, especially Morgagni. From the presence of these numerous and often large concretions arose the disease called "calculous phthisis," admitted by all the writers of the sixteenth and seventeenth centuries, and that Bayle restored to honor in 1810 by the publication of his great work on phthisis.

The ancient authors believed the calculous masses found in the lungs were due to the inhalation of dust, but Laennec believed them to be formed during the healing of tubercles, and that nature had been too extravagant in the deposition of the lime salts. Broussais considered them to be due to the degeneration of tubercles. In one hundred autopsies performed on old people Roger found concretions in fifty-one cases, mostly at the apex. Riellet and Barthez, in two hundred and sixty-five autopsies on tuberculous children, found them twenty-one times. These "lung stones" may, by what Laennec called "a secondary crop" of tubercles around them, be found in a cavity communicating with a bronchus. Subsequently these calcareous bodies—lung stones, as they are sometimes called—may be expectorated (Osler).

The lymphatic glands of the lungs are intimately connected with the bronchi, and where hypertrophied they may encroach on their calibre, and when inflamed they may become adherent to their walls, and thus cause perforation. The different phases of this process have been followed out by Barthez and Riellet (*Med. des Enfants*). In one case a bronchus was perforated by a small cretaceous gland. Situated directly behind the right bronchus was a gland about the size of a small pea, and containing two tubercles entirely calcified and about as large as millet seeds. At this spot the cartilage was eroded and the mucous membrane ulcerated, but to a less extent than the cartilage. A calculus contained in a lymphatic gland may, so it appears, find its way into a bronchus. *Circa divisionem trachea primam vomicæ adhaerenti parti ejus posticæ includebater calculus acutus et aspes, pisi magnitudine.*

The concretions found in the bronchi of tuberculous origin have been of variable volume up to the size of a hazel-nut; the form has been round or irregular; the structure porous; their consistence variable, some being easily crushed between the fingers, others as hard as stone. The color may vary from the whiteness of chalk to a dirty yellow. Microscopical examination, after previous treatment with muriatic acid,

shows no distinct structures. Chemical analysis proves them to be composed principally of carbonate and phosphate of lime.

Morbid ossification has been encountered in the lungs as in most of the other organs, and believed to be due to the result of chronic inflammation, and not to senility. Luschka found ossified places in the lungs of a man, aged fifty-four years, who died of chronic Bright's disease. Rokitansky attributed numerous attacks of pneumonia to the presence of two osseous masses in the lung tissue. Foerster, in Virchow's *Archiv*, showed that a lung stone he examined was made up of lung tissue ossified. The passage of these osteoids into the interior of a bronchus by a process of ulceration is probable. Dalmas said he saw these bodies "striking into the bronchi," but perhaps these were only cretaceous lymph glands.

Leroy gives the history of a case in a woman, aged forty-three years, who developed symptoms of pleurisy and effusion on the left side. At the autopsy the left pleural cavity was partly filled with a purulent exudate. A concretion was found in a dilated bronchus which communicated with the pleural cavity by a fistulous tract. No evidence could be found in the lung tissue that the concretion had formed in it, and both lungs were otherwise perfectly normal.

The following case (Leroy) is more happy in its termination, but is not complete, as the French wit said no case could be without a post-mortem. A doctor in active practice in the country began to have an obstinate cough, hectic fever, loss of flesh, and occasional hæmoptysis. There were moist râles and dull percussion sounds under the right clavicle. He continued to go from bad to worse, when one day in a fit of coughing he expectorated two small concretions that looked like the malleus and incus. The health was gradually re-established after this, and seven years later he was perfectly well.

It has been observed that during long-continued irritation the cartilages may become ossified in the non-dilated as well as in dilated bronchi. Later, these ossified portions may become detached from the bronchial walls and lodged in the bronchus. Leroy gives the long history of a case in which symptoms of an abscess existed at the base of the right lung; in a bronchiole close to this cavity three concretions were found, which on examination proved to be true bone or *ossified cartilage*.

As we have stated, most of the ancient writers believed the concretions found in the bronchi were formed there by inspired dust. Morgagni believed they were formed in the bronchi, and relates an instance in which a patient coughed up a concretion "which had its origin in the extremities of the small bronchi and the prolongation of the windpipe; it had the form of the place it occupied; it was oblong, cylindrical, slight, and was covered with little branches."

It has been said that inspissated mucus could act as a nucleus for the formation of a concretion in a bronchus, the lime salts being deposited on it. Laennec gives a note by Andral: "In the lungs of a man, aged sixty years, who had never had any signs of lung disease I found several calculi, hard as stones, with some branches like the renal stones. These calculi probably had their origin in the bronchial ramifications. The lung tissue was perfectly healthy everywhere."

Leroy extracts from *Bull. et Mém. de la Soc. des Hôp. de Paris*, 1865, p. 6, a most interesting case, particularly with reference to the original case given here, but unfortunately our space will only permit of a short reference to it.

A man, aged thirty-four years, had pneumonia six years ago, since which he has had cough and deeply-felt pain in the right side of the chest. During the past two years every month or two he developed symptoms of a *vomica*, when he had to quit work and remain in bed. There would be great pain, fever, profuse fetid purulent sputum. This condition would last some fifteen days, when he would be able to return to his work. After a particularly severe one of these attacks he had a sudden, brisk hæmoptysis, and while coughing and spitting out the blood he heard a concretion strike loudly in the basin. After this the man completely regained his health. There was no history of his having got a foreign body in his lung, and his work was not dusty. The concretion was not chalky. It was compact and hard, but neither cartilaginous nor bony in its structure. It weighed forty-seven centigrammes and had about a dozen rootlets.

In *Les Bull. Soc. Anat.*, t. xxviii., p. 88, et *Bull. Soc. Méd. Hôp.*, p. 9, Leroy finds the following as showing concretions do form in the bronchus: A man had spitting of blood and signs of pneumonia in the left side. At the autopsy no signs of tubercles were found, and the bronchi were dilated. One of these contained a small chalky mass, which was branched, and around it the lung tissue was pneumonic; the mucous membrane of the tube was ulcerated, but the tube was not perforated. There was a purulent effusion in the pleura.

In the diagnosis of a bronchial concretion we have no specific signs to aid us. The symptoms would lead to the diagnosis of the existence of a phthisical cavity or perhaps to bronchiectasis, "which results from the presence of a foreign body in the air tubes" (Osler). The absence of tubercle bacilli in the sputum will eliminate tuberculosis, and perhaps the Röntgen rays might aid in discovering a concretion of sufficient magnitude.

From the foregoing cases it is seen that ordinarily the patient is troubled for a greater or less length of time with cough, first dry, then followed by mucous or mucopurulent sputum. The physical signs may be those of a cavity, and the general symptoms those of hectic (septic) fever. Dyspnœa, pain, and hemorrhage do not help us. The

anomalous character of the symptoms could lead us to suspect their nature. Leroy gives as an example of the diagnostic difficulties in these cases of bronchial concretions the following case reported by M. Barth (*Bull. Soc. Méd. des Hôp.*, 1865, p. 9:

A young woman had a very serious and obstinate bronchitis, though auscultation gave no signs of its being of a tuberculous character. At a certain period of the disease the expectoration became very considerable, and one morning a concretion as large as a grape-seed was spit up; a little later a second concretion was expectorated. After this the health was rapidly restored. The concretions were believed to have ulcerated their way through the bronchial walls from the lymphatic glands.

Without going into profitless speculation as to the origin of the calculus thrown off in the original case reported, the opinion was formed that the concretion had probably been latent in the bronchus for a long time, and that in some way it was disturbed and caused to act as an irritant, making a *locus minor resistenciæ*, and in the light of modern bacteriology the habitat of the body and its adjoining tissue became infected; hence the bronchitis, the pneumonia, the tissue necrosis, and the happy termination in the freeing and expelling of the body. In this case, previous to the onset of the attack of bronchitis, the man had been employed at some unusually heavy and arduous work, shovelling coal in a coal-lighter. The unaccustomed exertion entailed in the performance of such extremely heavy labor could perhaps have disturbed the quiescent concretion.

PROGRESSIVE MUSCULAR DYSTROPHY, WITH THE REPORT OF AN AUTOPSY.[1]

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MORE than twelve years have passed since one (Sachs) of the present writers discussed the subject of progressive muscular atrophy before the American Neurological Association. During this period innumerable contributions have appeared relating to the amyotrophies and the dystrophies, yet many of the same questions which puzzled us at that time remain unsolved. It is generally assumed that the amyotrophies

[1] Read before the New York Neurological Society, March 5, 1901.

can be easily differentiated—at least clinically—from the progressive primary myopathies; the spinal forms are developed late in life; there is in them no distinct hereditary influence; the wasting begins in the upper extremities, and rarely in the lower; hypertrophy is the exception, fibrillary twitchings the rule, while the electrical changes are more or less pronounced, often leading to a complete reaction of degeneration. Contrast with this the early onset of the myopathies, the distinct hereditary and family taint in the victim of this affliction in whom the first symptoms appear in the lower extremities and not in the upper; hypertrophy is the rule, fibrillary twitchings the exception, while the electrical conditions show no very marked departure from the normal.

The simplicity of this clinical scheme is disturbed by the fact that a sufficient number of cases of spinal muscular disease have been reported in adults as well as in children, showing a distinct family taint. Some hypertrophied muscular fibres have been made out in these same forms, and often the electrical changes have been noted to be very slight indeed; on the other hand, the fibrillary twitchings and the reaction of degeneration which have been thought to be thoroughly characteristic of the amyotrophies have also been noted in the cases of primary myopathy. In view of the difficulty of making a proper clinical differentiation, an opinion uttered five years ago may be repeated here, that "it is wiser to be guided by the general agreement of symptoms than by any one single symptom." But even this will not help us altogether out of the difficulty, for Friedel Pick, for instance, reported only lately a case which he had diagnosticated as a spinal form of progressive muscular atrophy, but on post-mortem examination not a single trace of spinal disease could be made out. Similar experiences have led Placzek to decide that, in view of the many difficulties besetting the differentiation between the various groups of progressive muscular atrophy, it would be wiser not to attempt to distinguish between them and to regard all of them as belonging to a single morbid entity. It is doubtful, however, whether it is necessary at the present moment to accept this extreme view.

Remembering, also, that it is uncertain whether the peroneal form of progressive muscular atrophy should be classified with the amyotrophies, or whether it is properly designated as a progressive neural atrophy, we are bound to admit that the clinical distinction between the various amyotrophies and dystrophies is not as firmly established as we could wish. As between a progressive neural atrophy and an amyotrophy, the neuron theory will help us to account for the lack of a sharp line of demarcation in the symptomatology, and will also help to explain the difference in the anatomical findings of the cases of progressive neural atrophy hitherto reported, some authors finding distinct

changes in the spinal cord and others denying the existence of these, and maintaining that the peroneal form is due solely to a peripheral nerve lesion. There is, indeed, a strong possibility that in some instances the distal end of the neuron, and in others the central end, may be more distinctly diseased; the clinical manifestations of such lesions would, however, differ but very little. One would, nevertheless, be justified in looking for a marked differentiation between the primary myopathies and the amyotrophies, unless it could be shown that even in the myopathies it is the distal end of the neuron which is first affected. Such evidence has not been furnished in abundance, although a lady physician, Dr. Saccara-Tulbure, has claimed to have found distinct changes in the terminal endings of the nerves in the muscles in a case of pseudohypertrophy. She insists that, by methods which she employed, she could make out a distinct diminution of the tufts and fibres in the terminal muscular plates. It is a little unfortunate that the case in which she has found this was not an entirely typical case of pseudohypertrophy, although it was allied in all probability to the group of progressive dystrophies.

In former years much stress was laid upon the histological structure of the muscles in the two groups of cases, and we were inclined to believe that the simple examination of a piece of muscle removed during life or post-mortem would help to establish a positive diagnosis. In the muscles removed from dystrophic patients hypertrophied fibres were abundantly in evidence, or else fibres that had undergone simple atrophy. In amyotrophies hypertrophied fibres were not found, and there was a marked degeneration, not a simple atrophy, of the muscular tissue. But since that time it has been shown by a number of authors that hypertrophied fibres occur in purely spinal affections, in infantile spinal paralysis, and in syringomyelia, for instance; while, on the other hand, Hoffman maintained, with good reasons, that simple atrophy of the muscular fibre occurs in the spinal forms of progressive muscular atrophy. Moreover, the question is still open for discussion whether or not the gray matter of the cord is affected in the primary dystrophies, and, if affected, whether such changes are primary or secondary. Erb, Pick, Kahler, Heubner, and others have reported changes in the gray spinal matter in cases of primary dystrophy. Erb and Strümpell regard these changes as secondary, but in the case of Strümpell, Hoffman, whose authority in this matter could hardly be questioned, doubts whether the case upon which the argument was founded was a primary dystrophy at all. In the attempt to reconcile conflicting views, Erb has interjected the further proposition that all the progressive myopathies (whether or not there be anatomical evidence of central nervous disease) are of neuropathic origin and due to *functional* disturbance of the trophic centres, and that the results of

such disturbance of the trophic centres would be evident first in the parts furthest removed from these centres, namely, in the muscular tissue.

It is difficult to prove or to deny the truth of such speculation, but it would seem to us that if purely functional changes are to account for such a wide-spread disease as is progressive myopathy some slight structural change might be made out in the gray matter of the spinal cord in patients who have survived for many years the onset of the disease. The first case, upon which we report in detail, will offer valuable evidence in this direction, and the value of it is increased by the fact that the latest staining methods and the best technique have been employed by Dr. Brooks in his examination of the nerves and muscular system. We propose to show by the records of this case that in a progressive muscular dystrophy, lasting over fifteen years, the anatomical findings in the spinal cord were practically negative. In this special case the disease began as a typical pseudohypertrophy, but as the boy was one of a family of three in whom the second brother developed a characteristic juvenile form of progressive muscular atrophy, and another brother also developed a muscular pseudohypertrophy, we may infer that the negative findings of this case have a special value with regard to all the forms of progressive muscular dystrophy.

The clinical history of the patient, J. K., cannot be given with fullest details, because, unfortunately, some of the earlier records of the case were not retained at the Montefiore Home, into which institution the boy was admitted on December 15, 1889. But the loss of the earlier records matters little, for the boy was under observation for a period of nearly eleven years at the home, and during these eleven years his condition changed but very little. He died of pneumonia March 15, 1900. At the time of his admission he was thirteen years of age; was born in Russia; his parents were alive and well at that time. A younger brother had suffered from a similar affliction. This boy was also transferred to the home, where he died in 1898 of cardiac disease. He presented the clinical symptoms of Erb's juvenile type. Unfortunately, no autopsy was obtained. A third brother has the ordinary form of pseudohypertrophy. His present condition is unknown to us. There was no history of any other taint in our patient's family.[1] Early in life J. K.'s parents noticed constant movements of the head and of the eyes. He was practically well until the age of ten years, when he had a fall and is said to have broken his left leg. He was confined to bed a few weeks, recovered from the injury, and could walk about until one year later, when he fell again, and for a second time is supposed to have broken his leg at the same point. He recovered from the second fall, but could not walk about as well as formerly. One year before admission, at the age of twelve years, he was stricken with typhoid fever; was confined to his bed for two weeks, and when he arose from his sick bed and attempted to walk his parents noticed that

[1] The pictures of these three brothers will be found in Sachs' Nervous Diseases of Children, pp. 422, 423, 424.

he could not get about. A few weeks after this he could not walk at all. The hypertrophied calves were very striking at the first examination, but they had not attracted the attention of the parents. Barring a slight progression in the atrophies of the muscles, the boy's condition changed so little that it may be summarized as follows: The head was large, undergoing constant rotary movements, congenital nystagmus, macroglossia, and thick speech, all of which may be taken as distinct stigmata of degeneration, as has been noted in other cases of pseudo-hypertrophy. His general intelligence was fair. He was instructed while at the Home, and became a very assiduous reader. There was a marked atrophy of all the muscles at the shoulder girdle, the upper arm and forearm, the muscles of the hand alone preserving anything like a normal configuration, but the grasp of the hand was almost *nil*. He presented typical "loose" shoulders. The deep spinal muscles were intensely atrophied and caused distortion of the spinal column and trunk. The thigh muscles were atrophied, the calves remaining large and tough to the very end, although they had become diminished in size during the last few years. Both feet were club-shaped and in extreme valgus position. The exact measurements are given in the pathological report, and mention of them is therefore omitted from this clinical statement. During the last five or six years of his life the boy was only able to use his facial muscles and to hold his head erect when sitting in a chair. He was totally unable to raise his head from the pillow or to perform a single movement of the trunk or of the limbs. He had to be lifted about, as it were, *en masse*, and was a dead weight in the truest sense of the term. With the few exceptions above noted, the entire muscular system was useless. The case was, therefore, an extreme illustration of a progressive muscular dystrophy of a pseudo-hypertrophic type. The reflexes were, of course, absent. In the earlier years there was some response to the faradic current, which disappeared as the atrophy increased. The vesical and rectal reflexes remained normal to the very end. The general health was good, and if it had not been for the onset of pneumonia the boy might have lived for many years.

Pathological Report. The necropsy was performed twenty-four hours after death.

"The body is that of a small male. It shows great general deformity, and is much smaller than normal for a subject of this age, being about the size usual in a boy of fourteen or fifteen years. The head is, however, of natural size, and consequently appears proportionately very large to the small and wasted trunk and extremities.

"The face is asymmetrical, the right side is puffy, flabby, and has a paretic appearance not unlike that seen in myxœdema; the left side is much more natural, and has a more intelligent look. There is a scanty, fine growth of brown beard and moustache. The left eye and eyebrow are elevated, and the pupils are dilated irregularly. The nose is broad, short, and the alæ nasi are very thick and heavy. The oral cavity is very broad. The teeth are irregularly placed and badly shaped. The tongue is large, broad, and very thick, though proportionately thin at the tip, which is somewhat deviated to the left. The ears are large and project well from the head. The Darwinian tubercles are, however, small. The general expression of the features is dull and passive, and somewhat simulates the facial aspect of acromegalia.

"The forehead slopes back sharply and the integument covering it shows several deep transverse folds. The entire head is vertically elongated, so that the occipital protuberance projects but a short distance posterior to the line of the neck. The scalp is covered with a thick growth of fine brown hair, darker in color than the hair on the face.

"The neck is small as compared to the head, but is about proportionate to the trunk. The sternocleidomastoid muscles are contracted, and the head is rotated slightly to the right.

"Both arms and forearms are small, and their musculature is soft and flabby as well as scant in amount. The hands are small, delicate, and well formed. The fingers are medium flexed. The entire upper extremities may be said to be generally atrophied, the condition being much less marked in the hands than elsewhere; it seems like a passive atrophy, the hands being probably less atrophied on account of their greater functional activity.

"The thorax is greatly deformed. The entire cage is deviated laterally to the left, so that the right side projects sharply forward, the lower border returning to jut out toward the right. This deformity is caused by a marked latero-posterior curvature of the spinal column, the greatest deviation of which is to the left.

"The curvature of the spinal column causes a compensatory deviation of the pelvis to the right, so that its median line lies about 6 cm. to the right of the axis of the thorax and in the line of the head and neck. The blade of the right iliac bone is also deflected to the left, so that its crest reaches beyond the median line of the pelvis. The left ilium is correspondingly but much less deviated.

"The external genitals are small, but are normally formed.

"The lower extremities are very short, even as compared to the thorax. Both are flexed at the knees, and when extended the right measures, from the great trochanter to the plantar surface of the arch, 71.5 cm., and the same measurement on the left gives 73 cm. The musculature of the lower extremities is scant, that of the left side being of somewhat greater volume than that of the right. The musculature of the thighs and buttocks is small in amount, but is unusually solid to palpation. The calves are of small volume; at the point of greatest diameter the left measures 27 cm. and the right 25 cm. The peculiarity of these muscles is the curious wood-like, inelastic impression which they give to the palpating hand; in this they contrast very markedly with the soft and flabby muscles (though equally atrophied) of the upper extremities and neck. The feet are inverted so that the plantar surfaces are almost parallel. Both tendo Achilles are so contracted by the retraction of the calf muscles that the toes are drawn nearly to the axis of the shanks. The feet are thickened laterally, but are short, very firm and solid, resisting pitting almost completely, although they have the appearance of being œdematous. The circumference of the right foot at the arch is 26.4 cm., and the same point on the left gives a diameter of 23.8 cm. The length of the left foot from the tip of the os calcis to the end of the great toe is 18 cm., and that of the right foot 17.2 cm. The relative atrophy of the calf muscles is very apparent when one compares these measurements with those of the calves. The toes are all sharply flexed as though from contracture of the plantar muscles and tendons.

"The thoracic musculature is very small in amount, but that of the abdomen is more abundant, and the muscles here are of more natural color and consistency, though they show in lesser degree the changes which are most extreme in the greatly wasted pectoral muscles in which muscle tissue has apparently been substituted by strands of fibrous-appearing tissue of unnatural firmness and inelasticity.

"The lungs are collapsed, permitting a large exposed præcordia.

"Both pleural cavities are free and normal.

"The pericardial sac contains 20 c.c. of clear, light-colored serum. The epicardium shows a few areas of opalescence, which are most numerous, thickest, and largest over the left ventricle. The left ventricle of the heart is widely distended, while the right ventricle is collapsed. The aorta has a diameter of 5 cm. just above the sinuses. The aortic valves are normal, but there is a relative insufficiency of the mitral segments. The valves of the right heart are normal throughout. Both auricles contain small post-mortem clots, but the ventricles are for the most part empty. The myocardium shows a great diffuse fibroid infiltration, which is most extensive in the wall of the left ventricle, where it is distinctly patchy in distribution. The intima of the aorta and the endocardium is slightly thickened in places, but hardly enough so to be considered a pathological state. The coronary arteries are normal in appearance. Weight of heart, thirteen ounces.

"The aortic arch throughout the ascending and transverse portion shows a moderate arterio-sclerosis, which is not, however, continued into the carotids or other large branches.

"The lungs are moderately congested. There are several calcified tubercles in both apices, and a few scattering tubercles throughout the substance of both lungs, but in every instance the tubercles are surrounded by a dense, usually calcified capsule, and all are apparently quiescent. The tip of the right lower lobe is involved in an extensive area of recent bronchopneumonia, and the bronchi distributed to this area are filled with a thick, yellowish pus.

"The tongue is very large. It measures from tip to the base of the epiglottis 14.5 cm., and is 7.6 cm. broad at its widest part. It is 3.6 cm. in thickness.

"The lymph nodules of the posterior dorsum are enlarged and moderately injected.

"The lymphatic tissue of the pharynx is generally hypertrophied It is otherwise normal.

"The thyroid gland is of usual size. The thymus body still persists and is proportionately as large in size as in the child. The tissue seems to be of the same structure as is found in the infant.

"The lymph nodes of the cervical and bronchial chains are moderately enlarged and pigmented.

"The diaphragm is relaxed, it presents no abnormality in texture or thickness, and the smooth muscle tissue making it up seems to be perfectly normal.

"The liver is small and somewhat irregular in shape. Its structure is normal.

"The spleen is enlarged to about three times its usual size. Its tissue is deep mahogany in color, firm and congested. The Malpighian bodies are apparent to the unaided eye, and the connective tissue of its septa seems to be somewhat increased. There are two spherical super-

numerary splenic nodules, each 2 cm. in diameter, near the hilus of the organ. Weight of spleen, six ounces.

"The omentum and mesentery are scant in adipose. The mesentery lymph nodes are moderately enlarged, and are very firm in consistence, apparently from fibroid increase.

"The stomach is small. It contains 200 c.c. of normal appearing chyme. The mucous membrane seems to be natural, and there is no apparent disease of the muscle coats.

"The duodenum, jejunum, and ileum are entirely natural. The amount of fecal content is normal.

"The vermiform appendix is folded into a small coil and is bound to the posterior surface of the caput coli by old bands of adhesion. There are no evidences of acute inflammatory process.

"The large intestine is normal.[1]

"The pancreas is large; it is very firm, light in color, and of apparently normal structure.

"The adrenal bodies are small, the cortex is light yellow in color, and the medulla a deep red. There is no gross lesion.

"The kidneys are of about the usual size, but the left organ is considerably deformed, apparently from the pressure exerted on it by the deviation of the spinal column. The markings are fairly distinct. The capsules are not adherent, and the organs are quite natural in appearance save for a general but moderate congestion of the bloodvessels. United weight of kidneys, six ounces.

"The ureters are normal.

"The urinary bladder contains about 500 c.c. of normal urine. The mucosa of the cavity is normal, and there is no apparent disease of the muscular tissue.

"The prostate gland, urethra, testicles, and penis are all small, but are in perfectly normal condition.

"The large abdominal bloodvessels follow the deformities of the spinal column in their course. The intima of each shows a moderate amount of arterio-sclerosis, the process being less apparent in the smaller trunks.

"The skullcap is thin and quite deeply arched, though the fossæ on both sides are rather shallow, the arching being, however, somewhat more free on the left side. The configuration of the base of the cranium is normal, and it presents no deformity.

"The dural sinuses contain a small quantity of dark, partly clotted blood.

"The dura is normal. The pia-arachnoid is somewhat congested, but is not thickened and not adherent.

"The brain is of good size, is fairly symmetrical and its tissue is firm. The bloodvessels of the base are in normal condition, and those throughout the brain tissue are but moderately congested. The convolutions of the cerebrum are not particularly abundant, but they are quite typical, symmetrical, well formed, and ample, while the sulci are moderately deep. The cortical layer of gray matter is thick and regular. No gross lesion is present in any part of the encephalon.

"The pituitary gland is of usual size and apparently of normal structure.

[1] Muscular alterations in the coats of the intestinal tract were especially sought for but the findings were entirely negative.

"The membranes of the spinal cord are negative.

"The posterior root ganglia are normal to the unaided eye; they are large and firm, but not abnormally so.

"On inspection, and on section, the various levels of the spinal cord seem to be normal; it is not abnormally firm, and the appearance of the white and gray matter to the unaided eye shows no peculiarity, and the differentiation is normally distinct.

"Examination of many of the peripheral nerve trunks from various parts of the body showed no gross abnormality in any, and the amount of perineural fat seemed in each case to be neither increased nor diminished.

"The inguinal lymph nodes are considerably enlarged, the enlargement being apparently of a fibroid character.

"No lesion of the smooth involuntary muscles in any part of the body was found.

"Nearly all the voluntary muscles showed fibroid substitution to a greater or less degree, varying greatly in amount but apparently always of the same character."

The following muscles were especially examined:

"The psoas muscles show an extensive fibrosis.

"The muscles of the back are universally of small volume, and all show extensive fibroid replacement, being light in color, very firm, and inelastic. In places there is an extensive replacement with a firm fibroid yellow fat. The fibres of the latissimus dorsi can be identified in a few places, but are mostly unrecognizable. The trapezii are very extensively invaded. The rectus capitis group are perhaps somewhat less involved, but are very firm, retracted, and inelastic. Even the muscles of the scalp show extensive fibroid changes perfectly evident to the unaided eye, but the most extreme of all the lesions of the muscle tissue is to be found in the muscles of the calves where the tissue cannot be recognized as muscle, being wholly transformed into a diffuse solid mass of firm yellowish-white tissue, apparently made up of mingled areolar tissue and fat, and in which the substituted muscle blends so completely with the structure of the tendons that it is impossible to say where muscle leaves off and where tendon begins. This tissue is of almost wood-like density. All the tendons and fasciæ of this region are much contracted.

"Cause of Death: Bronchopneumonia and fibroid myocarditis, complicated by progressive atrophy and fibrosis of the voluntary skeletal muscles."

As the tissues were removed from the body they were immediately placed in various hardening fluids. After they had become sufficiently fixed and hardened they were placed in 80 per cent. alcohol for preservation. The microscopical study was begun about five months after the time of death.[1]

These examinations were conducted with care and in great detail; as the findings were, however, largely negative they will not be given in full, but only the points of greatest importance will be considered.

The most pronounced changes of the voluntary skeletal muscles were found in the muscles of the calves. Sections of the soleus and of the

[1] We are indebted to Dr. Isador Unger, U. S. A., for the preparations of the tissues of this case.

gastrocnemii showed an almost complete substitution of muscle elements by a firm, dense growth of areolar connective tissue, everywhere of an adult form and with no evidences of recent cell growth or fibre production. In places the areolar tissue had become transformed into a very dense variety of adipose, characterized by the small size of the fat cells and the relatively great amount of large and densely arranged connective tissue fibres. Occasionally remnants of voluntary muscle fibres were found, but they were rare, very small, usually wholly without nuclei, and cross-striation was indistinct or absent.

Sections of the right psoas muscle showed similar conditions qualitatively, but here muscle fibres were much more abundant and more natural in appearance. The hyperplastic areolar connective tissue was of the same type as that found in the calf muscles, and there were also no evidences of recent connective tissue proliferation. Of the muscle fibres that remained few could be called natural; most of them were either larger or smaller than the normal, varying from small to very large, while other fibres were contracted at one point and expanded at another. Cross-striation was evident in most fibres, both large and small, and some showed this feature even more plainly than normal. Many of the cells enclosed a fine brown pigment, and in such cases there was generally more or less granular disintegration of the sarcoplasm with a corresponding indistinctness of cross-striation. Still other fibres showed the sarcoplasm mostly transformed into large fat-containing globules which were still invested by sarcolemma. Nuclei were present in most of the cells, and when present they were usually in greater number than normal and were to be seen in rows of from three to eight, just beneath the sarcolemma or extending deep into the sarcoplasm. This was especially shown in those cells which presented the more advanced stages of degeneration without fatty disintegration. Some of the supernumerary nuclei exhibited karyokinetic-like figures; usually these were atypical, and not true mitotic figures. These evidences of progressing nuclear multiplication were much less frequent than is generally found in cases of acute or progressive muscular atrophy.

The changes in all the other voluntary muscles were of a precisely similar character, varying only in the extent of the alterations. Next to the calf muscles the pectoral groups seemed most involved, though here the amount of connective tissue hyperplasia seemed to be proportionately less as compared to the muscular atrophy. The lesions were least marked in the occipital muscles, where the amount of connective tissue hyperplasia was less and the degenerative manifestations of the muscle fibres not so far advanced. Complete degeneration of the sarcoplasm was rare in these muscles, but nuclear proliferation and hypertrophic fibres were frequent. Karyokinetic-like figures were common in the chromatin of the cell nuclei, much more so than in any of the other muscles. While the atrophic changes in the fibres of these muscles were less advanced, yet no evidences of active proliferation of the cells of the connective tissue could be made out. In a few places there were collections of small round cells, situated in the region of arterioles or capillaries where they probably represented a certain degree of true inflammatory process.

Examination of the various distributions of smooth muscle tissue failed to show any evidences of degeneration or of hyperplasia of the connective tissue forming its framework, beyond a certain small amount

of proliferation of the connective tissue surrounding the bloodvessels, which was found to be a general condition throughout all the tissues. Of the involuntary muscles so examined the chief were the diaphragm, the muscular tunics of the urinary bladder, of the stomach and intestines, and the muscularis of the medium and small sized arteries.

Microscopical examination of the heart muscle showed a much less degree of interstitial increase than the gross appearances led one to expect. The wall of the left ventricle exhibited a moderate augmentation of interstitium, the hyperplastic tissue being entirely of an adult type and being most abundant about the vessels. The papillary muscles showed proportionately a much greater interstitial increase. The cardiac muscle cells were found everywhere to be in a very natural condition, and, notwithstanding the apparent degeneration noted in the gross examination, there was no atrophy of the cells, no abnormal pigmentation, and no evidences of abnormal nuclear activities.

Sections of the lung taken from the pneumonic areas showed the ordinary picture of pneumonic consolidation. Sections from the uninvolved portions showed a moderate degree of emphysema, a general congestion of the capillaries, and a considerable anthracotic deposit in the lymph spaces about the air sacs. The larger vessels of the lung tissue showed an increase of the perivascular interstitium, but without any evidences of a recent process. Where the sections included small bronchi, their mucosa was found considerably congested.

Sections of the persistent thymus body demonstrated it to be made up of diffuse adenoid tissue with occasional spheres of condensation. Hassalls' corpuscles were rather less numerous than is usually the case as found in early childhood.

Sections of the thyroid gland showed no deviation from the normal.

Examination of sections of the pituitary body showed acidophylic cells numerous in the glandular portion, but no distinct lesion was found in either glandular or neural lobe.

The vessels of the adrenal bodies were found considerably engorged, and there was a moderate increase in the connective tissue framework of the glands. Examination showed no abnormality of the parenchyma.

Examination of the liver demonstrated a considerable general dilatation of the portal capillaries with congestion. There was a moderate degree of connective tissue increase in the capsule of Glisson, which was, however, devoid of all evidences of recent proliferation. Some of the liver cells contained more than the usual amount of pigment, but the fat content of the liver cells was not above the normal. The hepatic parenchyma may be said to have been practically in a natural condition.

Sections of the spleen showed a considerable degree of congestion, especially of the blood spaces. There was a general thickening of the capsule and of the septa. The Malpighian nodules were well differentiated and normally abundant. There was a large deposit of coarse, black pigment in the cytoplasm of the liver cells, free in the tissue, and distributed throughout the capsule and septa diffusely and in the connective tissue cells.

There was also an extensive anthracotic pigmentation of the lymph nodes of the neck, and to a lesser degree the same was present in the nodes of the mesentery and of the abdominal chains. The connective tissue increase in the lymph nodes was very small, and the moderate

hypertrophy of the bodies seems to have been due to congestion and to pigmentation.

The pancreas was found normal, save for a slight increase in the amount of connective tissue.

The stomach and intestine showed nothing abnormal. These sections were examined particularly for alterations in the smooth muscle.

Sections of the kidney showed a considerable increase in the amount of connective tissue, the hyperplastic tissue being found both in the adult and in the actively proliferating forms, though areas showing recent growth were found only occasionally, the chief hypertrophy being in the adult form. There was a moderate general congestion of the vessels. The parenchyma in places showed a few granular and degenerated cells, but was as nearly normal as can be found in the adult human kidney under ordinary conditions.

Sections of the urinary bladder showed no abnormality.

The bloodvessels as found through the various tissues universally showed an increase in the amount of connective tissue surrounding them and uniting them to the adjacent structure. Few exhibited sub-endothelial hyperplasia to any considerable degree, and the inner coat of the intima was found almost always in a normal condition. No areas of perivascular infiltration were found, except as noted in the occipital muscles, and even in the sections of the extensively diseased muscles the blood channels seemed to be in a comparatively normal condition. No evidences of new vessel formation were found, and apparently the connective hyperplasia in the diseased muscles took place, largely independent of any marked vascular alterations.

Numerous peripheral nerves were examined, both in sections of the diseased muscles and as separate trunks, especially prepared to show fibre degeneration if that lesion were present. No fibres were found which showed degeneration of appreciable extent as demonstrated by the methods utilized (Müller's fluid fixation with Pal-Weigert staining; Marchi's fixation; alcohol and formalin hardening with hæmatoxylin and eosin, and Van Gieson's stains). Some of the larger trunks, as the crurals and sciatics, showed a considerable increase in the amount of endoneurium, but close observation showed this hyperplasia to be clearly of a perivascular type, originating about the small arterioles of the trunk. Sections of the atrophied muscles, which included nerve trunks, showed a perceptibly less amount of connective tissue hyperplasia in the make-up of the nerves than in the surrounding tissue.

Sections of the cœliac sympathetic ganglia fixed in alcohol and stained after the method of Nissl showed absolutely no changes which could be looked upon as degenerative, although occasionally a few cells did show more than the ordinary amount of brown pigment deposited in the cytoplasm.

Unfortunately, only a few posterior root ganglia were properly preserved for study by the Nissl method. Those which were so fixed universally showed identical lesions. These lesions consisted in a shrinkage of the ganglion cells, in many cases similar to that produced occasionally by fixing agents, but here the irregular perilymphatic spaces enclosing the shrunken cells were found filled in with the proliferating capsular cells. This would seem to indicate that cell shrinkage had taken place long before death, and that it was distinctly a pathological process. By far the greater number of ganglion cells showed a natural

condition of the Nissl bodies, but occasional cells, especially the shrunken ones, were found in which an almost complete chromotolysis was present; chromolitic changes of moderate degree were still more frequently found. Chromophylia was present in a few instances, and was usually peripheral, rarely involving the entire cell cytoplasm. Nuclear changes were wholly wanting. The amount of coarse, brown pigment normally present in these ganglion cells seemed to be augmented in a few cases, and an identical pigment was occasionally found free in the stroma of the ganglion in more than natural amounts. Apparently the connective tissue stroma of the ganglia had been considerably increased, but there were no indications that this process was still advancing.

The histological examination of the spinal cord was thoroughly made, each separate segment being studied, usually by several methods, in order that different types of tract or cellular degenerations might be sought for.

The connective tissue throughout the entire cord was found to be increased, the hyperplasia in each part being strictly of a perivascular origin and of but a moderate degree. The bloodvessels of the cord were universally congested, but this congestion was apparently of a hypostatic nature. There were no indications of an inflammatory process in any part, except in the perivascular increase of connective tissue, which was obviously of a chronic character and probably but a localized manifestation of the general process.

Degenerated tracts were absent in every part of the cord. This conclusion was reached after careful study, utilizing the stain of Pal-Weigert and corroborating these findings by Marchi's method. Occasional isolated degenerated fibres were found (if we may depend absolutely on the methods employed), and these were usually in the columns of Gall or Burdach, but nothing in the nature of a systemic degeneration was indicated in any level.

Morphological changes in the ganglion cells of the spinal cord were entirely absent, and sections stained with Nissl blue demonstrated that the cytoplasmic and nuclear structure of the ganglion cells, in so far as can be determined by this method, were also for the greater part normal. Occasional ganglion cells presenting different types of degenerations as demonstrated by this method were, however, found, and it is necessary that these be considered in more or less detail.

In the cervical region the ganglion cells of the anterior horns showed in a few instances a slight nuclear eccentricity; the most common chromatophilic change in the cells of this region was a clumping of the chromatic substance into several larger and darker staining bodies while the intervening network and the remaining plaques, though they could be seen, refused the stain to a more or less degree, apparently because these chromatic elements were very finely divided. Cells were found very rarely in which there was no apparent reaction to the dye; usually, however, plaques could be faintly seen even in these cells. The dendrites universally retained their response to the stain, though in some instances the chromatic bodies seemed to have been broken up into very finely divided granules. The posterior horn cells showed degenerative alterations with equal rarity, and where change was noted it usually consisted of a finely granular disintegration of the plaques, which resulted in a more or less diffuse staining of the cytoplasm. This was limited to no particular group or variety of cells. Occasionally the

achromatic elements stained to a slight degree. No nuclear changes were noted in these cells.

In the dorsal portion of the cord peripheral chromotolysis was infrequent, but the most common alteration was a perinuclear chromotolysis with a clumping of the chromatic substance in the peripheral portions of the cytoplasm. In those cells where the chromatic substance was more or less clumped the intervening unstained plaques were frequently made up of very finely divided chromatic granules. The network in the faintly stained archochromes was always very evident, even though it had refused the characteristic stain. Nearly all of the cells which showed these degenerative alterations still showed a normal arrangement of the chromatic elements of the dendrites. Eccentricity of the nucleus was found much more commonly in the dorsal cord than in the cervical segments. Not infrequently the cells which showed perinuclear chromotolysis presented a more or less marked nuclear chromotolysis as well, in which case the outlines of the nucleus blended very closely with the net of the cytoplasm. Alterations in the cells of the posterior horns were more infrequent than in the anterior horn cells.

In the lumbar cord chromolitic changes were still more rare. The most common lesion consisted in a finely granular subdivision of the plaques with a corresponding loss of staining affinity; usually this process did not involve the entire cytoplasm. Perinuclear chromotolysis was much less frequent than in the dorsal segments. A few of the lumbar cells showed an unusual amount of brown pigment collected about the nucleus.

As the brain had been hardened for the purpose of tract study, the ganglion cells of those centres could not be studied by the more recent methods. It seems very improbable that any lesion would have been found had the study been made.

The chief lesions which bear on the general condition may be summarized as follows:

Extensive atrophy, which affected nearly all the voluntary skeletal muscles, and was confined to these muscles.

Production of diffuse areolar connective tissue and adipose tissue which replaced the substance of the atrophied muscle.

Slight general perivascular connective tissue hyperplasia.

Moderate interstitial myocarditis.

Extensive degenerative changes in a few of the cells of the posterior root ganglia.

Rare and irregular types of cytoplasmic alterations without morphological change in the ganglion cells of the spinal cord.

1. Atrophy of voluntary muscles seems to have affected nearly all distributions of this tissue, though in markedly variable degree. Both history and pathological examination indicate that this process first began or was at least most rapid in the muscles of the calves.

As to the character of this process, in so far as can be determined by histological examination, it differs in no way from the atrophies which result in many widely different conditions, as from section of nerve

trunks, disease of the descending tracts, or even from injuries affecting the muscle tissue direct. It is highly improbable that there are any specific modes by which voluntary muscle fibres degenerate, as has been intimated by some authors.

The process may differ in rapidity or location, but follows on certain well-defined lines, and, as in Wallerian degeneration, is probably wholly unaffected by its etiology, excepting in degree and in reparative efforts. Everything points to a slow progression of the proces,s which was doubtless inaugurated in but a limited distribution of the tissue, subsequently affecting all voluntary striated muscle.

That the atrophy of the muscle fibres preceded connective tissue hyperplasia is indicated by the conditions found in the less extensively involved muscles, where obviously the process is most recent. As an example we may cite the picture presented in the occipital muscle.

Complete absence of the changes in the smooth muscle shows that the disease process was not a general one of all muscle tissue, but was strictly localized in the voluntary muscle distribution.

Since the muscle cells of the heart showed no variation although there was a moderate interstitial increase in this viscus, we also exclude striated involuntary muscle from the involved tissues.

2. We do not look upon the connective tissue hyperplasia which took place in the process of substitution of the atrophied tissue as a primary evidence or manifestation of the disease. Everything points to the fact that the connective tissue increase was but an example of the universal function of this tissue, which by its growth fills in and replaces tissue of any form when it becomes extensively diseased or is removed.

The fact that this tissue in its growth followed natural tendencies, passing from a proliferating, semi-embryological form to a normal adult type, shows that the growth was not of itself pathological, but that it was directly allied to the normal conservative process familiarly known and described in scar formation.

3. Perivascular connective tissue hyperplasia though general was of slight degree. Of such extent it is found more commonly than not, and it certainly cannot be considered a lesion typical of the disease or produced by it. It cannot be a causative factor, or the atrophic changes found only in the skeletal muscles would also have been found in other structures.

4. Probably the moderate degree of interstitial myocarditis was associated with the connective tissue hyperplasia of the bloodvessels. Possibly it was a primary process incited by overaction of the heart or by some other and unknown cause. There is no reason why we should look upon this as in any way connected with the factors producing atrophy of the voluntary muscles.

5. We certainly must admit that the changes found in the posterior

root ganglia are of great significance. Actual morphological alterations in the cells, as well as extensive cytoplasmic changes, cannot be attributed solely to the terminal infective process or to post-mortem decomposition. It is not so clear, however, that these bear a direct relation to the atrophy of the voluntary muscles. The fact that so few of these cells were involved while the extent of the muscular atrophy was general must exclude these lesions from our consideration as primary. That secondary alterations in the ganglia might result from primary disease of the muscle seems more probable. This last is our interpretation of the degenerations, which are then secondary in nature and perhaps dependent on death or extensive disease of certain of the terminal portions of the neuron—that is, of the sensory nerve endings of the involved tissues.

A process similar to this results after amputation, especially if, as Schaffer points out, the subject be young. The cell degenerations in amputation develop very slowly, as is also the case in this condition. Undoubtedly changes in the peripheral sensory fibres precede the ganglionic degenerations. They were not observed in this case, probably because the methods now at our command are insufficient to demonstrate slight changes in single fibres of the nerve bundles.

6. We naturally look for changes in the spinal cord in processes of this nature which are progressive and so symmetrical in location as to at once attract attention to the probability of a central lesion. The majority of previous researches in this disease have, however, tended to show the absence of such lesions. It seemed, nevertheless, possible that these negative results may have been brought about by deficient or faulty methods of research, and for this reason it is important that, as newer methods of investigation are elaborated, the ground be gone over again in the hope that more perfect methods may give more certain results.

On this account our chief study in this case has been directed to the spinal cord. In summary, our results are these: There are no evidences of tract disease. The occasional isolated degenerated fibres found in the posterior columns are probably ascending central branches of the degenerated spinal ganglion cells, though it seems that most of the ganglion cells affected must have been such as have their central fibre terminate in the gray matter of the cord. Were this not the case we certainly should have found something more in the nature of a systemic degeneration of the ascending tracts. The cytoplasmic degenerations of the ganglion cells of the spinal cord are rare and of such a character as might result from post-mortem changes or from the terminal infection which was the direct cause of death. The ganglion cell changes do not appear to us sufficient or of such character as might be expected were the process a secondary one following the long standing muscular

atrophy; though it seems probable that secondary degenerations of these cells might eventually result as in amputation.

For lack of a better explanation we are inclined to account for the somewhat striking chromophylic changes in certain of the cells of the dorsal segments as due to nutritive disturbances particularly affecting this portion of the cord, following the extensive and rapidly progressing curvature of that portion of the spinal column.

The chief inference to be drawn from the negative findings of this case is that the spinal cord of a patient who had suffered for over fifteen years from a distinct form of progressive muscular dystrophy did not present any evidence tending to show that the wasting of the muscles could be attributed to disease of the gray or other matter of the spinal cord. Whoever wishes to maintain Erb's theory is, of course, at liberty to do so, but it should be regarded as pure speculation. For the present there seems to be sufficient warrant to maintain the distinction between the amyotrophies and the myopathies. It is difficult, however, to account for this latter group of family afflictions unless we are satisfied simply to state that there must in these cases be some defect in the embryonal structure which does not permit of a satisfactory development of the muscular system. That the disease represents a primary affection of the muscular fibre may be taken for granted, and that the fatty degeneration of the muscular fibre is secondary to the hypertrophy and atrophy of the muscular tissue.

There are two questions which it seems wise to raise for discussion:

First, are all these cases of progressive dystrophy primary diseases of the muscular system, and may not some of them be the local expression of a universal lipomatosis? Only recently one of us had occasion to examine a boy who presented all the clinical symptoms of a pseudohypertrophy; but with the development of the pseudohypertrophy there was also developed an enormous universal lipomatosis, so that it was difficult to state whether the boy's inability to use his limbs was due to this general accumulation of fat or to a purely muscular affection. This same boy presented other distinct stigmata of degeneration, such as defective mental development and a marked chronic hydrocephalus. The occurrence of stigmata of degeneration in so many of these cases of progressive muscular dystrophy would lead one to suspect that these diseases should be classified broadly among degenerative family disorders; and possibly we may reach a better understanding of these family afflictions if we depart from the purely anatomical lines and do not regard them merely as affections of the muscular or neural systems.

Secondly, the question arises whether these muscular dystrophies are necessarily progressive. Even though they may be predestined, as it were, to develop some form of progressive wasting of the muscles, is it not possible that the tendency to progressive deterioration of the

muscular system may cease either spontaneously or may be checked by treatment directed to this end? A few rather noteworthy experiences which have been accumulated during the past seventeen years have led us to be more hopeful of the future of these cases, and in these experiences—few as they are—there is sufficient encouragement to urge that in every case of progressive muscular dystrophy the possibility of great improvement by systematic exercise should be kept in mind at the very start; and there is little doubt that if earnest efforts in this direction be made at an early period, better results may be obtained than has fallen to the lot of these unfortunate patients hitherto.

The most marked instance we have to record is that of a young woman, now twenty-five years of age, who first came under our notice at the age of ten and one-half years, in September, 1886, and whose condition was reported in the *Journal of Nervous and Mental Disease* and in the *New York Medical Journal* of 1888. The history taken at that time was briefly this:

The mother was forty-seven years of age, the father ten years older; the latter has since died of sarcoma. The patient was the youngest of all the children, and was born fourteen and a half years after the next oldest sister. During the first ten months of her life there was no trouble. The parents lived next to the Brooklyn Theatre at the time of the fire, and were compelled to take the child out in that cold December night. The parents lost all their worldly possessions at that time. The mother became greatly agitated, and, nursing the child at the time, she supposed that the trouble which was developed later on was naturally due to this sad occurrence. At first the child lost flesh, and was not able to stand until she was one and a half years old. She did well until the age of six, when the first difficulty in walking was noticed, and when the mother observed that the calves were growing larger; also, that both thighs were out of proportion to the rest of the body. At that time the faradic response was absolutely normal in all the muscles and nerves of the lower extremities. In January, 1887, it was noticed that the electrical condition remained normal, and that she had great difficulty in getting up and down from a chair or in rising from the floor. The flexors of the legs and thighs were particularly weak. In 1888 it was noted in addition that the calves and anterior thigh muscles were much hypertrophied; that the sternal portion of the cleidomastoid was distinctly atrophied; that the hand muscles were very thin; that she assumed the characteristic position in rising from the floor, presented a waddling gait, and that both feet were in valgus position; the arms seemed lean and long; all the muscles contracted properly to the faradic current, except the interossei of the hand and the vasti in the thighs.

The patient did not present herself again for the following eleven years, and since that time has remained continuously under observation. She had developed into womanhood and became a kindergarten teacher; and although she had experienced and still does experience some difficulty in going up and down stairs, and particularly in getting in and out of cars, she has been able to perform all her duties and is practically well, though much annoyed by the size of her hips and thighs,

and the circumference of her waist. She attributes much of her well-being to the sytematic exercise which she has been carrying on for years, and to the calisthenic movements which her calling necessitates her doing with children every day of her life. At present she merely exhibits some slight weakness of the orbicularis of the right side and of the deep spinal muscles. If placed on the floor and asked to rise she climbs up upon herself in typical fashion. The grasp of her hands is weak, but aside from this and the difficulty in climbing, and the slight waddling gait, she has little to complain of. Her intellectual development is surely up to the average. The size of her limbs may be inferred from the following measurements: Right thigh, 69 cm.; left thigh, 63 cm.; right calf, 36 cm.; left calf, 36½ cm.; measurement around hips, 112 cm.; waist measurement, 71 cm. (twenty-eight inches). The strength of the limbs is not in proportion to the size of the muscles, and it is fair to assume that the muscles are still in a condition of hypertrophy. There is no unusual lipomatosis, for above the waist line she is thin rather than stout. There is still evidence of a weakness and atrophy of the muscles around the shoulder girdle, the pectoralis showing slight wasting. The young woman has been encouraged to keep up muscular exercise, but not beyond the point of easy fatigue, for it is evident that any excess in this direction would be followed by atrophy. According to present indications there is no reason why the girl should not continue in good health for many years to come; and whether or not there was a spontaneous cessation of the tendency to hypertrophy and atrophy, the case has run an exceptionally favorable course, and as such is worth recording.

The second case was one which Dr. Wiener reported a number of years ago from my (Sachs') clinic.[1]

This case was exhibited before this Society some years ago. A few points of the history worth noting are that the patient at the time of the first examination, in 1893, was twenty-three years of age. Since May, 1892, he had been complaining of pains following upon active exercise; had experienced special difficulty in walking up and down stairs. The weakness of the muscles was first evidenced in the legs, then in the back, then in the hips, and, finally, in the shoulders and neck. The examination revealed marked atrophy of the muscles of the shoulders, back, hips, and thighs, as well as of the facial muscles. There was marked lordosis in the lower dorsal and upper lumbar region, He had loose shoulders and the typical gait of pseudohypertrophy. A microscopical examination of a piece of muscle removed from the deltoid revealed atrophic fibres and a proliferation of the muscle nuclei; also, some cellular infiltration in the interstitial tissue. By means of systematic gymnastic exercises, which Dr. Wiener directed, a marked improvement was brought about in the patient, so that he was able to go about his occupation, and, as I understand, has become a very expert bicycle rider. In Dr. Wiener's article a special note was made that the only muscles which did not improve were those of the face, and these had not been subjected to special exercise.

[1] Neurologisches Centralblatt, 1896, No. 9.

In clinical and private practice during the past few years other cases of pseudohypertrophy have been examined. and each and every one of them has been subjected to systematic exercise; and while it is still too early to report definitely upon them, it is at least safe to infer that the progressive deterioration of the muscular system can be checked, for a time at any rate, by active exercise. After all, the life of every part of the system is maintained by compelling it to perform, as far as possible, its natural physiological function. There is some reason to think that by this simple method much can be accomplished in the progressive myopathies. At all events, it would be a satisfaction to feel that a small number of the patients afflicted with progressive muscular disease may be rescued from the sad fate that awaits so many of them.

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THE MALARIA OF THE TROPICS.

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In the light of our recent knowledge of the subject, malaria may be defined as a specific infectious fever, characterized symptomatically by the following stages: Rigor, hyperpyrexia, and diaphoresis, occurring periodically; pathologically by certain changes in the elements composing the blood, and invariably by the presence in the blood of the plasmodium of Laveran; changes in the internal organs, and in the bone marrow.

Etiology. The predisposing causes are tropical climates, summer seasons, low marsh lands, dampness, decaying vegetable matter, and the prevalence of a certain species of mosquito—the Anopheles claviger.[1]

The exciting cause is the plasmodium of Laveran, which gains admission into the blood by the bite of the mosquito.

[1] It is probable that several other species of mosquito act as the intermediate host.

Pathology. The blood. In almost all cases of the disease, and invariably in those of long standing, and particularly in those presenting the more grave clinical pictures, we find the total bulk of the blood more or less reduced.

The red corpuscles are always reduced in number; in the milder cases, produced by the benign parasites, the reduction averages from 5 to 8 per cent.; this reduction in the number of red cells is progressive, both with the number of attacks and with the severity of the disease; for instance, in some of the pernicious cases we find that they do not average more than a million to the c.mm., and are frequently less than this.

The hæmoglobin is also reduced to a marked degree, usually out of all proportion to the loss in the number of red cells, the reduction ranging from 10 or 20 to 50 per cent. (Manson), the red corpuscles not attacked by the parasite being quite pale in color.

The shape and size of the red cells attacked by the parasite depends upon the type of the invading parasite. In the case of the benign quartan the red corpuscle retains its shape and size; in that of the benign tertian the red cell is enlarged; it is only in the blood of those patients suffering from a malignant type of the disease that we find the small, crenated, warped, and distorted red corpuscles known as the "brassy body."

In the malignant cases the white cells are frequently increased, and in some types of the disease markedly so.

Free pigment floating in the blood plasma can frequently be found, but it is usually observed within a leucocyte or a dead parasite. The amount of pigment depends upon the type and severity of the attack. In advanced cases of malignant malaria it is always quite abundant.

The Internal Organs. The spleen. This organ is always enlarged, frequently reaching an enormous size. It is more or less pigmented, and in cases of long standing may be almost black in color. Its glandular structure is usually found to have undergone fatty degeneration, and its bloodvessels are filled with débris of broken-down cells, plasmodiæ, and pigment.

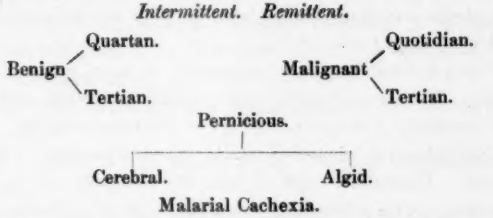
The liver is usually more or less enlarged, congested, and pigmented.

The lungs are frequently pigmented, and the heart in cases of long standing usually presents more or less evidence of fatty degeneration.

The substance of the brain is usually of a leaden hue, and its vessels are frequently filled with pigment and numerous parasites. The small vessels are sometimes the seat of parasitical emboli.

The bone marrow of the spongy bones is congested and degenerated, and is frequently heavily pigmented.

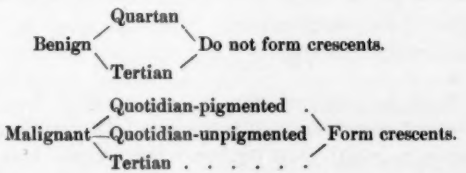
CLINICAL CLASSIFICATION OF MALARIAL FEVERS.



The tendency in all malarial infection is for the fever to intermit, and it is only through accident that we meet with cases which remit. The manner in which this occurs is as follows: Instead of the quartan, tertian, or quotidian parasites in a selected case maturing simultaneously numbers of them mature and sporulate before their brothers, thus giving rise to various typical cases of a remittent character.

But because we see so many cases of remittent malaria clinically, especially in tropical countries, it has been deemed wise to adhere to the name in this classification.

Malaria classified according to an examination of the blood would be arranged somewhat differently, viz., according to the species of the plasmodium producing a typical phase of the disease. Dr. Manson's classification with reference to the blood examination is as follows:



Intermittent Type.

Premonitory Stage. It frequently happens that for a day or two prior to a malarial attack the patient will complain of malaise, headache, vague muscular pain, anorexia, and constipation. These symptoms are suddenly superseded by the

Stage of rigor, which is ushered in by a more or less severe chill. The features become blue and pinched, the teeth chatter, and the patient complains of extreme cold, and asks for all the covering he can obtain. Vomiting is frequently a severe and very distressing symptom. If the temperature is taken it will be found to be several degrees above normal. This stage lasts about an hour, when it is followed by the

Stage of hyperpyrexia, in which the temperature suddenly rises to 104° or 105°, sometimes reaching 106° or even more. The patient's face is

now flushed, the eyes injected, the pulse full, rapid, and bounding. The skin is dry and hot, headache is severe, and vomiting frequent. These symptoms continue from three to four hours, and are in turn superseded by those of the

Stage of diaphoresis, in which the patient is in a profuse, drenching perspiration. The temperature now rapidly subsides, and all other symptoms gradually disappear. In a few hours, except for a feeling of lassitude, the patient is apparently well. The temperature will probably be subnormal. This afebrile period lasts from twenty-four to forty-eight or seventy-two hours, depending upon the type of parasite productive of the symptoms, when, if not treated, they usually reappear, although spontaneous recovery sometimes occurs. This periodical return of the symptoms may continue for some time, and if the parasite is not removed from the blood some atypical form of the disease may ensue.

Diagnosis. The presence of the plasmodium of Laveran in the blood will prevent mistakes in diagnosis.

Prognosis. Favorable as to the duration of the attack.

Treatment. Rest in bed upon a liquid diet, 30 grammes of magnesium sulphate given at once, then, as Manson advises, wait for the appearance of sweating, when 0.648 grammes of quinine should be administered. During the next twelve hours 1.324 grammes of the drug should be given. Upon the three succeeding days 2 grammes of the drug should be used, when, if no recurrence of the disease occurs, the dose should be cut down to 0.324 grammes t. i. d., and continued over a period of five or six days.

The Remittent Phase.

This form of malarial infection is not typical of the disease, but its frequent clinical occurrence makes a general description necessary.

The manner in which it is produced has already been described, so it can readily be understood that an infection produced by any one of the five malarial parasites may result in an atypical case, in which the fever will be remittent in character.

It may begin as a pure intermittent, and gradually become remittent. This usually occurs in cases of benign infection, or it may remit from the start, which is frequently observed in cases of malignant infection.

With the exception of the character of the fever, the symptoms, on the whole, to a considerable degree, resemble those observed in cases of the intermittent type, but are not so pronounced, the three stages of cold, heat, and free sweating not being so readily separated, or they may be altogether absent, or which would be more true not recognizable, the patient simply complaining of feeling somewhat worse at a certain period of the twenty-four hours, and it is this periodicity so character-

istic of malarial infection which must be looked for if mistakes in diagnosis are to be avoided.

The tendency in all cases of remittent infection is for the disease to be prolonged; especially is this true where the infecting parasite is malignant in character.

Diagnosis. To avoid error a blood examination should be made as soon as possible.

Prognosis. This is not nearly so favorable as in the cases of intermittent type.

Treatment. Rest in bed and a liquid diet. The administration of a brisk saline purge and the early and immediate use of quinine in large doses.

Commence with one gramme and follow this in the succeeding twenty-four hours by three grammes; then continue to give 2.6 gramme a day until the patient is thoroughly cinchonized, after which the dose may gradually be reduced, and finally discontinued, depending upon the microscopical examination of the blood.

After prolonged treatment, should the organism persist in the blood, arsenic should be pushed to its physiological limit, at the same time administering iron in the shape of tr. ferri chlor.

Should the organism still persist, a change of climate should be advised.

Malignant Types.

Although the fever associated with malignant malarial infection is usually remittent in character, still we do see well-marked cases of typical quotidian or tertian intermittent, but they are comparatively rare and usually sooner or later assume the characteristics of the remittent phase of the disease.

The malignant cases differ clinically from the benign, in that they are usually of longer duration, relapses are frequent, at intervals of from ten to fifteen days they show a decided tendency to develop cachectic symptoms, and in addition to the more pronounced headache, anorexia, loss of flesh and energy, the patient complains of neuralgic pains in different parts of the body, great mental depression, the skin and scleræ are stained a deep yellow, pigmented patches appear upon the body, hemorrhages occur from the mucous membranes and beneath the skin, the latter frequently giving rise to well-marked purpuric conditions.

During the later stages of the disease the heart becomes weak, rapid, and irregular, in some cases tachycardia being very pronounced.

The bowels may be constipated, or a bilious diarrhœa may be associated with the other clinical features.

It is in these cases that pernicious symptoms suddenly develop, pro-

ducing death in a few hours, or, at times, leaving the patient a paralytic invalid, or, less frequently, partially or totally blind.

The prognosis is very unfavorable if the patient cannot obtain change of climate, and then many of them remain permanent invalids.

The treatment embraces climatic, hygienic, and medicinal features. Medically the treatment should follow the lines already considered under the treatment of the remittent phase of the disease. In severe cases, or those which persist after prolonged and careful treatment, a change of climate should be advised; a dry, moderately cold, sunny locality should be selected if possible. The patient's clothing should be at all times warm, as any sudden chilling will be productive of bad results.

Pernicious Attacks.

These may occur during any case of malarial infection, but are more apt to be associated with the malignant cases. They may be divided into two classes, viz., cerebral and algid.

The cerebral attacks are more or less varied in character, the most common seizure being one associated with hyperpyrexia and coma, the temperature suddenly jumping up to 108° or 110° or even 112°; or with a temperature of 104° coma suddenly develops, which may disappear with the appearance of sweating, or the patient may die of exhaustion.

The attack may result in permanent paralysis, and in some cases a temporary or permanent blindness may ensue.

The algid attacks are associated with greatly lowered surface temperature, shock, and collapse. They may be accompanied by gastric symptoms, persistent vomiting, with gastric pain and distress, or they may be of intestinal type, sometimes closely resembling cholera, or, in other cases, there may be the bloody mucous stool of dysentery, all these conditions tending to end in fatal syncope.

The cerebral cases with high fever should be treated with ice baths or an ice pack, and about six grammes of quinine should be administered as quickly as possible, two grammes being given by the mouth, two by the rectum, and two hypodermically.

The algid forms require in addition to quinine active stimulation, and should be kept absolutely quiet, as the least exertion may induce fatal syncope.

Hæmoglobinuric Fever (Black-water Fever).

This form of malarial infection, first observed and reported by French naval surgeons serving on the African station, and since observed in America, some parts of India, and Southern China, seems not to exist in the Philippines, or at least if cases have been met with they have not been reported.

This is an exceedingly fatal variety of infection. According to Manson, one out of every three or four cases ending in death, and those cases in which recovery takes place remain intensely anæmic and frequently develop seriously diseased kidneys. It usually occurs in patients who have had several prior attacks of intermittent or remittent fever, the disease generally beginning in much the same way. After a short time the patient complains of pain in the loins and over the region of the liver, which soon becomes severe.

Gastric symptoms are usually pronounced, incessant vomiting and great pain are frequently encountered. The bowels are usually constipated, but bilious diarrhœa is sometimes a marked symptom. The skin and scleræ are always stained a deep yellow. The urine becomes very dark, the coloring gradually growing more pronounced until it is almost black. The fever may be intermittent in character, occurring periodically every twenty-four, forty-eight, or seventy-two hours, or it may assume a remittent character or become irregular.

As the disease advances pernicious symptoms may arise at any time. They may be either cerebral or algid in form, death ensuing in a few hours; or the disease may take a favorable turn, the symptoms gradually disappearing, the urine slowly regaining its normal color, and the patient emerging from the attack in an intensely anæmic condition.

The urine should be carefully examined from time to time, as death from acute Bright's disease frequently occurs three or four weeks after all symptoms of the original disease have disappeared, or the kidneys may become the seat of chronic parenchymatous or interstitial nephritis.

Prognosis. Very unfavorable.

Treatment. Absolute rest in bed, and if possible an absolute milk diet should be insisted upon.

If the microscope should reveal the malarial parasite in the blood the treatment should be that previously mentioned in connection with remittent fever.

If constipated, 0.324 grammes or 0.648 grammes of calomel should be given, followed by 30 grammes of magnesium sulphate.

Upon the first sign of kidney insufficiency, diuretics should be freely administered, and hot flaxseed poultices applied to the loins. Should suppression with uræmic symptoms intervene, pilocarpine should be given hypodermically, and the patient placed in a hot pack.

Special Remittent Fevers.

Bilious Remittent. This fever is frequently met with in malarious localities, the characteristic fever being associated with marked gastric symptoms, sometimes the bowels are constipated, but more frequently bilious diarrhœa is present.

Remittent fever is frequently associated with typhoidal symptoms, a low muttering delirium with great prostration being observed.

These fevers, in addition to the usual malarial treatment, require special attention given their respective symptoms.

Malarial Cachexia.

A chronic condition resulting from severe or repeated malarial infection, characterized by pronounced anæmia with its associated symptoms, and usually by an intense yellow staining of the skin.

The patient is emaciated, the skin usually of a deep yellow hue.

Great muscular weakness is a pronounced symptom. Gastric distress in the shape of various dyspeptic conditions is common. The heart is weak and rapid, sometimes giving rise to a pronounced tachycardia, the least exertion resulting in varying degrees of dyspnœa.

Peripheral multiple neuritis is frequently met with, the superficial reflexes diminished or even abolished.

Headache and neuralgia, occurring periodically, are almost constant symptoms.

Mental depression, sometimes verging on melancholia, is frequently present.

Hemorrhages from the mucous membranes, and beneath the skin, with varying degrees of purpura are seen.

Inflammation of the spleen and liver, with their associated symptoms, are common. In the case of the former enormous enlargements of that organ are frequently observed.

Microscopical examination of the blood will reveal anæmia, varying from a slight reduction of the red cells, to a condition of the blood closely resembling that of pernicious anæmia; or the white cells may be increased, giving rise to pronounced leucocytosis.

This condition renders the patient particularly liable to the contraction of many serious and fatal diseases, such as pulmonary tuberculosis, catarrhal pneumonia, typhoid fever, and dysentery, and to the development of chronic disease of the kidneys and fatty degeneration of the heart.

A change of climate should be advised. The patient should be warmly clad, and the diet should be nutritious and stimulating. A methodical superfeeding should be carried out.

The medical treatment should consist in administration of such drugs as arsenic, iron, quinine, and strychnine.

REVIEWS.

A Practical Treatise on Genito-urinary and Venereal Diseases and Syphilis. By Robert W. Taylor, A.M., M.D., Clinical Professor of Venereal Diseases at the College of Physicians and Surgeons (Columbia University), New York; Surgeon to Bellevue Hospital, and Consulting Surgeon to City (Charity) Hospital, New York. Second edition. One volume. Pp. 722, with 138 illustrations and 27 plates, in colors and monochrome. Philadelphia and New York: Lea Brothers & Co., 1900.

The profession has for years regarded Dr. Taylor as a leader in this subject, and has learned to trust in the matureness of his judgment and the wisdom of his advice. It is, therefore, with pleasure that we open this new volume, knowing that the contents will have the touch of the master's hand. The previous edition appeared in 1895, consisted of a thousand pages, and was justly considered a standard for books on that subject. In the preface of the present volume the author states that he has endeavored to present an up-to-date, practical, and compact treatise. As a result this volume is nearly one-third smaller than the last, and several subjects are treated so briefly and concisely as to be of little service to any except the genito-urinary expert; and yet with all the brevity the writer's views are summed up clearly in a few well-chosen words, and we are forced to believe that an extended consideration of surgical diseases of the kidneys, bladder, and prostate seemed to him out of place in a book of this character. Aside from this there is very much to praise in the work. The description of gonorrhœa and its treatment is excellent, and his forcible remarks on facts are timely and just. He believes that gonorrhœa in 80 or 90 per cent. of cases invades the whole urethra, and that only exceptionally is it limited to the anterior portion. Such a statement is undoubtedly true of dispensary cases, where the poor are unwilling or too ignorant to properly follow the treatment recommended, but when applied to private practice we would feel chagrined if posterior urethritis so often supervened. Under gonorrhœal rheumatism he states as a golden rule that the inflammation in the urethra must be cured, since it is the source and origin of the disease. If the hydroarthrosis in this complication does not speedily subside after the application of blisters, he advocates aspiration and irrigation of the joint. It is to be hoped that this teaching will have many disciples. His remarks on prostatic hypertrophy are excellent, and a clear conception is given when palliative treatment should cease and operative treatment begin. In hydrocele he prefers Von Bergmann's operation of resection of the sac, but does not mention Doyen's procedure, which accomplishes the same results in perhaps an easier and quicker manner.

The book closes with a series of chapters on syphilis, which are classical. Great emphasis is laid on the fact that the proper time to begin

systematic medication in syphilis is the date at which general manifestations show themselves—namely, as soon as the generalized rash appears together with the manifold symptoms of the secondary period. Few exceptions to this rule present themselves. The various forms of treatment are clearly detailed, and concise instructions given on the course to be pursued.

The book is singularly free from pages of prescriptions, a fault so common in works of this character. In all matters relating to treatment, both surgical and medical, the author has aimed at presenting wholesome, conservative, and practical directions, and to accomplish this in such a condensed form as the present volume shows a high order of ability and labor. We can unhesitatingly recommend this book to both students and practitioners. R. G. L C.

A PRACTICAL TREATISE ON SEXUAL DISORDERS OF THE MALE AND FEMALE. By ROBERT W. TAYLOR, A.M., M.D., Clinical Professor of Venereal Diseases at the College of Physicians and Surgeons (Columbia University), New York; Surgeon to Bellevue Hospital, and Consulting Surgeon to the City (Charity) Hospital, New York. Second edition. One volume. Pp. 438, with 91 illustrations and 13 plates, in colors and monochrome. Philadelphia and New York: Lea Brothers & Co.

THE appearance of a second edition of Dr. Taylor's book on this subject is good evidence that the first edition was well received by the profession, and satisfactorily filled a needed want. The book is a little larger than its predecessor, but still of a very convenient size. The text has been thoroughly revised, the black and white illustrations and also the colored plates are of high order, and freely illustrate the subject in hand. The chapters on the anatomy and physiology of the sexual apparatus have been much amplified, and the subject of sexual disorders in women is presented in a more comprehensive manner. Particular attention has been paid to the therapeutics, in the direction of clearness of statement and of practicability. Until recently the subject of sexual disorders has been treated in a rather loose and impractical manner, the work done being unsatisfactory and unscientific when viewed as a whole, and based to a certain extent on visionary theories. It has been Dr. Taylor's province to gather together the useful information that has been gained, and separate facts from fancies. As a result, we have a well-balanced volume on this highly important system. The underlying anatomical and physiological conditions have never been lost sight of in his description of diseases and symptoms, and the light of pathology has been thrown on the picture as fully as our present knowledge will warrant. The broad lines laid down by Dr. Taylor permit us to grasp comprehensively, on scientific and practical grounds, diseases which a few years ago were vaguely if at all understood, and as a result our treatment of such disorders is steadily progressing to a satisfactory basis.

The general practitioner is apt to have but few cases of these disorders, and his knowledge on the subject is usually correspondingly limited. Should he have occasion for such knowledge we can highly recommend to him Dr. Taylor's work. R. G. L C.

THE AMERICAN YEAR-BOOK OF MEDICINE AND SURGERY. Being a yearly digest of scientific progress and authoritative opinion in all branches of medicine and surgery, drawn from journals, monographs, and text-books of the leading American and foreign authors and investigators, collected and arranged with critical editorial comments, under the general editorial charge of GEORGE M. GOULD, M.D. In two volumes. Vol. I., Medicine. Vol. II., Surgery. Philadelphia and London: W. B. Saunders & Co., 1901.

DR. GOULD has adhered to the plan adopted last year of issuing this book in two volumes, one on medicine and the other devoted to surgical subjects. The volume on medicine is somewhat larger than that on surgery. The two volumes represent a very complete library of abstracts from current medical literature. These abstracts are wisely chosen, and the original matter has been carefully abstracted, the result being the production of a compend of modern medical literature in such shape as to be readily got at by those in pursuit of the literature of any particular subject. In the volume on surgery we would call particular attention to the section on general surgery. The subject is, of course, a large one, and we think that the method employed by the editors in the extraction of material of value from such a large field has resulted in a particularly useful epitome of the year's literature on the subject. In the volume on medicine the most notable sections are those on general medicine, by Drs. Stengel and Edsall, and on pathology and bacteriology, by Drs. Riesman and Kelly. In no branch of medicine have such extraordinarily important acquisitions to our knowledge been made as in pathology and bacteriology, and as upon these studies all clinical medicine must rest for its foundation, it is a great advantage for the busy practitioner to be able in a work of this character to find a condensation of the literature of the laboratory, with which it would otherwise be impossible for him to keep up; thus, in the study of yellow fever, cancer and malaria, accessions to our knowledge are made daily, possessing the greatest importance in the treatment of those conditions. Here the many articles which have been published during the past year by various laboratory workers are digested and the authoritative results indicated.

The handsome typography and make-up of the books are particularly to be commended. F. R. P.

THE MEDICAL NEWS POCKET FORMULARY. Containing 1700 prescriptions representing the latest and most approved methods of administering remedial agents. By E. QUIN THORNTON, M.D., Demonstrator of Therapeutics, Pharmacy, and Materia Medica in the Jefferson Medical College, Philadelphia. New (third) edition. Carefully revised to date of issue. In one wallet-shaped volume, strongly bound in leather, with pocket and pencil. Philadelphia and New York: Lea Brothers & Co., 1901.

WHILE the pocket formulary is in general a useful addition to the physicians' library, it of necessity has its limitation and dangers. For, after all, the practitioner of medicine must suit his combinations and doses to the individual case, and the tendency of a formulary is to encourage wholesale copying of prescriptions without a due study of

original causes. On the other hand, when the formulary is used as it should be, merely as a suggestion and help toward compounding the prescription, it is of great value, for often a physician may forget temporarily an appropriate drug or a happy combination, both of which may be supplied at short notice from the experiences of other men, most of them eminent in their different branches of the profession. The *Medical News Formulary* is one of the best that has appeared. It contains over seventeen hundred formulæ relating to the treatment of all diseases that the physician will encounter, and they are arranged in alphabetical order under the headings of the various diseases treated upon. And not only is the formula given alone, as is too often the case in such works, but there is appended to each a short note giving directions as to the administration of the remedy, the suitable diet for the case, and the indications calling for this particular line of treatment. Under each heading there are a number of different prescriptions, either for different phases of the same disease, or the treatments of different men. Care has been used to exhibit the drugs in a palatable form, and most of the remedies suggested are those whose action is well known and not fancy or untried preparations. In addition, there are tables of weights and measures, incompatibles, poisons and antidotes, and of dosage which are handy for ready reference. The volume is neatly bound in soft leather and of a convenient size to carry in the pocket; the text is clear and well printed on good paper. This little work has had a great deal of popularity in the past, as is evidenced by the rapid exhaustion of the two preceding editions, and as a number of new drugs and different combinations of old ones have been included in the present (third edition), it will undoubtedly be as much appreciated as the others. G. M. C.

Obstetric and Gynecologic Nursing. By E. P. Davis, A.M., M.D., Professor of Obstetrics in Jefferson Medical College, and of Obstetrics and Diseases of Infants in the Philadelphia Polyclinic. 12mo., 402 pages. Illustrated. Philadelphia and London: W. B. Saunders & Co., 1901.

This volume has been prepared as a text-book for obstetrical and gynecological nurses, and is dedicated to the training schools of the Jefferson and Philadelphia Hospitals. The teaching is clear and concise, at the same time conservative, and the style is excellent. Much stress is laid upon the importance of antisepsis and cleanliness in every duty which the nurse is called upon to perform, and the relations which should exist between the nurse and the patient and the nurse and the physician are clearly brought out. We are particularly pleased to note this advice to the obstetrical nurse: "If the physician requests her to tell him whether a laceration is present or not, she should respectfully decline, saying that she is not competent to make the examination." The book is well illustrated and is divided into two parts, the first being devoted to obstetrical and the second to gynecological nursing. The author's definition of gynecological nursing is open to question and will not be generally admitted: "This branch of nursing has to do with

those conditions of ill health in women in which the pelvic organs are concerned, but in which disease does not arise from pregnancy, parturition, or the puerperal state. As the majority of diseases among women are connected with pregnancy or parturition, it will be seen that the field of gynecological nursing is a narrow one. As much of the treatment employed is of a surgical nature, it is more a surgical specialty than a separate and distinct department of medicine."

As a rule, the trained gynecologist is better qualified than the obstetrician, both by experience and skill, to perform the secondary plastic operations and the abdominal operations necessitated by abortion and the accidents of pregnancy, and the gynecological nurse must be thoroughly trained for this work. She is also frequently called upon by the gynecologist to nurse patients who do not require surgical measures, and whose disorders are not due to disturbances incident to puberty, menstruation, and the menopause.

Careful and clear instruction is given for the care of the infant and the disorders which may arise. The appendix contains dietary formulæ and methods for the preparation of surgical supplies. J. B. S.

CLINICAL STUDIES IN VICE AND IN INSANITY. By GEORGE R. WILSON, M.D., Medical Superintendent, Mavisbank Asylum. Pp. 234. New York: MacMillan Company. Edinburgh: William F. Clay, 1899.

THE author divides his book into three parts, viz.: "1. Drunkenness and alcoholism considered as always a morbid affection of the purposive or self-directive functions of the mind, with special reference to the dynamics of *cerebral* processes."

"2. Types of alcoholists—twelve cases, some of them peculiar, considered especially from the point of view of the question, What kind of men and women become drunkards?"

"3. A clinical study of insanity from records of fifteen cases, with various digressions—the mental physician's point of view."

Writers have endeavored to make a distinction "between drunkenness, inebriety, alcoholism, dipsomania, mania a potu, delirium tremens, alcoholic insanity, alcoholic epilepsy, as if each were a disease by itself." These terms are often convenient, but the author makes an appeal for a recognition of the fact that all of these disordered conditions have a definite and fairly constant lesion running through them, the difference depending mainly "in the part of the nervous system or organ affected, the localization or diffusion of the lesion, the acuteness or intensity of it, and its complications," and suggests that a generic term as "alcoholism" should be used. Dr. Anstie has defined alcoholism to be "a disease of the general nervous system, induced by continued excesses in the use of alcoholic liquors." Victor Magnan and others have made contributions to the literature of alcoholism, not from the stand-point of the moralist, but founded upon the clinical experience of physicians. The term has come to have a medical significance, and the conditions described have a tangible recognition. A nomenclature or classification is always helpful if generally understood and accepted, and the author, at the risk of a new venture in this direction, would use a

descriptive adjective, as simple or chronic alcoholism; epileptic alcoholism; alcoholic insanity; delusional alcoholism, etc. As many of the morbid manifestations, both mental and physical, that belong to the excessive use of alcohol are also observed from the prolonged use of drugs, and we might add the abuse of drugs, it would seem there is a warrant for a supplemental nomenclature, so as to include cases of this character.

Admitting the difficulty which attends a study of the physiology of mind from a knowledge of the anatomy of the brain and of understanding the relation of symptoms of disease to pathological conditions, the author proceeds to present "a dynamical view of brain physiology," or the operation of nerve motion upon the dendritic portion of the brain, and attempts to lay a foundation of the pathology of drunkenness. It is not alleged that these views are founded upon clinical observations; and whether they are to be numbered among the many instances of indulgence in fruitless theoretical speculations about this subject, time and further consideration will show.

That prolonged indulgence in alcoholic dissipation results in pathological changes in the brain has been shown by Dr. H. J. Berkely and others. Although the connection between these abnormal conditions and the various nervous and mental manifestations that follow is not yet understood, they are sufficiently related to establish the existence of disease and to bring these cases within the domain of medical science and study. The author might have laid a stronger foundation for the title of his book if he had furnished a history of the published cases prior to the manifestation of pathological or mental changes. It will appear, as a rule, that the majority of cases of alcoholism give some indications of degeneration to a lower plane, which is one of the results of heredity, and some of the cases cited establish this fact. A lowered moral sense and absence of will-power are the usual accompaniments of vicious propensities, so that bad habits acquired by doing a thing over and over again are no longer resisted, and become easily fixed. Vicious habits may become by repetition as firmly established as those of decent living, so that the moral aspect of these cases cannot be wholly ignored in the diagnosis or in the after-management.

There will be a general agreement with all that the author proposes as to the means and importance of re-establishing the general health of alcoholics; but he omits the most important element of treatment, which is the power of detention of the patient, in the absence of which all efforts are futile. How this is to be exercised, or under what conditions it will be granted, cannot be discussed here. If given, it will be against the judgment of public officials; of lawyers who will look upon this step as a move to abolish a sacred privilege of the citizen, and if ever granted will be done grudgingly, and only after it has been demonstrated by medical experience to be an essential to successful treatment. There is no problem in morals, sociology, or in medicine which has been approached or considered in such a cowardly manner as the relation of the drink habit, vice and vicious habits to insanity. It becomes the medical and legal professions and all boards having to do with the incompetent classes in their various relations to give the various questions that are involved their fair and best consideration.

The number of applications made by alcoholics for admission to hospitals intended for the care of the insane, to sanitariums and so-called

"cures," often under the management of charlatans, is an evidence of their desire, as well as the victims of all habits, for relief. As a contribution from the large clinical experience of Dr. Wilson we welcome this book and all efforts to place the study of alcoholics and habit cases on a higher medical plane.

The cases of insanity reported have a value as showing the relation of vice and vicious habits to the production of this disease. It may be noted, also, that many of them bear indications of transmitted predisposition to depravity and disease, and to a degree illustrate what is often observed—that vice, crime, and alcohol are among the most prolific indirect causes of insanity. J. B. C.

ANATOMY IN ITS RELATION TO ART. An Exposition of the Bones and Muscles of the Human Body, with Especial Reference to their Influence upon its Actions and External Form. By GEORGE MCCLELLAN, M.D., author of "Regional Anatomy," etc. Illustrated by 338 original drawings and photographs made by the author expressly for the work. Published by the author, 1900.

DR. MCCLELLAN has again written and himself published a most sumptuous work on human anatomy. The scope of the present work is confined to the relationship of anatomy to art—a subject in which this acknowledged master of the science of human construction is peculiarly fitted to speak with authority and to present it with all the added force of delicate artistic appreciation, he himself being skilful in the arts of drawing and photography and familiar with the needs of an artist from many years of experience as a teacher of anatomy at the Pennsylvania Academy of the Fine Arts. The result, as embodied in this large quarto, fully justifies the expectation of his many appreciative pupils and friends in the medical profession. They would expect much from so competent a teacher, not only in technical completeness, but original presentation, and the product will sustain all tests.

The special features of the work are the studies of the skeleton in relation to the outline of the body and to the photograph of the model, care being exercised to secure (by great labor and trouble) skeletons and models which shall exactly correspond and be as nearly as possible perfect in proportions.

Next the individual parts of the bony framework are studied, and the distinctly original plan is pursued of sketching in the deepest muscles first, then in increasing series the other muscles, until the completed part is presented as a photograph of the model. This is different from the older books, since elsewhere the plan is followed of dissecting away the skin and gradually reaching the deeper structures. In the method employed by Dr. McClellan a clearer idea is secured of the relationships of the bones to the muscles, and also the method of reproduction used displays these parts in a diagrammatic fashion, giving the directions of the tendinous and ligamentous structures, but presenting most beautifully a quality of transparency to the limb or part analogous to an idealized fluoroscopic view.

Another striking feature of this book, also distinctly original, is the

systematic comparison, side by side, of certain of the best specimens of antique statuary and photographs of the living model. In this connection Dr. McClellan makes a great point of calling the attention of artists to the importance of holding constantly in mind that this work has to do with living anatomy in normal poise and not with crude dissections of the dead as they are tempted to do. Much of modern statuary exhibits this fault, and the consequence is an appearance of exaggeration and overtension marring grace and finish.

This is particularly well shown in the plates of the "Sandal Binder," the "Discobolus Thrower," the "Flying" and the "Resting Mercury," and the "Fighting Gladiator."

Photographs of the female figure are also shown in a number of customary and domestic poses, which bring out the beauty of outline in contrast to the skeleton in simple and reposeful attitudes. These, again, contrast charmingly and usefully with the male poses.

Some of the most valuable and original features of the book are Dr. McClellan's researches on the proportions of the human figure. He has taken pains to compare the observations of Vitruvius and others and verify them where they are warranted and to offer his own conclusions. This has involved a very large amount of study and labor, which forms an invaluable chapter at the close of the book and offers practical suggestions which will prove useful to the student of human proportion.

The construction of the book itself is unusually handsome, the text being printed in a dark gray and the plates upon a specially prepared paper, also in light tints and in a rich, warm gray, giving a delicacy to the proportions.

The depths and qualities of the photographs, too, are preserved to an unusual degree in reproduction. J. M. T.

MODERN SURGERY: GENERAL AND OPERATIVE. By JOHN CHALMERS DA COSTA, M.D., Professor of the Principles of Surgery and of Clinical Surgery, Jefferson Medical College, Philadelphia; Surgeon to the Philadelphia Hospital and to St. Joseph's Hospital, Philadelphia. With 493 illustrations. Third edition, revised and enlarged. Philadelphia and London: W. B. Saunders & Co., 1900.

THE appearance of this the third edition only two years after the second shows that the verdict of the profession has been favorable to the work.

It is not simply a reprint of previous editions, but the whole work has been most thoroughly revised. Each separate chapter seems to have been gone over and alterations and additions made. The total increase amounts to two hundred and six pages of new material, distributed pretty evenly throughout the volume. This, of course, brings the work well up to date and adds much to its previous value. The original aim, as stated by the author, was to present in clear terms fundamental principles, the chief operations, and the accepted methods of modern surgery, and to do this in a work which would stand between the complete but cumbrous text-books and the incomplete but concen-

trated compends. He was successful in accomplishing his purpose in the previous editions, and he has not departed from the lines then laid down in this edition. Subsequent issues, however, will demand pruning down if additional new matter is inserted. The work as it stands is an exceedingly useful one, both to the student and practitioner. The former is not confused with numberless details, while the latter will find in it almost everything he seeks for. The descriptions, while necessarily brief, still contain the essential points, together with many suggestions and hints that will be found of value. The work is very practical in character, and, while the most advanced procedures are referred to, a judicious conservatism has been exercised in those recommended, thus rendering it a safe guide for practice. G. G. D.

ORTHOPEDIC SURGERY. A HAND-BOOK. BY CHARLES BELL KEETLEY, F.R.C.S., Surgeon to the West London Hospital, etc. London: Smith, Elder & Co., 1900.

THIS is a medium-sized volume of 539 pages and 253 illustrations. The author states that the work was planned out nearly twenty years ago, and he publishes it as a statement of the views and as an analysis of the observations of a surgeon who has for twenty-two years devoted much time, thought, and labor to studying deformities and practising their treatment. Tuberculous diseases of the bones and joints, with the exception of spinal caries, are omitted, as are also ankyloses. They are reserved for appearance at a subsequent date in the form of an essay on the pathology and treatment of diseased and injured joints.

The book will be found to appeal both to the general practitioner who wishes to inform himself on the subject of orthopedics as well as to the specialist. It embraces chapters on knock-knees, bow-legs, rickets, various paralyses, cerebral and spinal; flat-foot, club-foot, hysteria in relation to orthopedics, wry-neck, deformities of the hands, fingers, and toes; congenital dislocations of the hip; the various diseases and deformities of the spine, acromegaly and a detailed description of the application and management of plaster-of-Paris and poroplastic felt. It is written in an excellent style. The descriptions and explanations are concise, clear, and readily understood. There is an absence of verbosity, involvement of sentences, and prolixity that will commend it to the busy man. At the commencement of each chapter is a synopsis of its contents, a half to three-quarters of a page being consumed. This could be condensed with advantage. The book is not a compilation of other people's work, neither is it a bold statement of the opinions of the writer. Contemporary literature has been studied and new procedures are explained and the author's views given as to their value. We judge that it is a fair presentation of modern British orthopedic surgery. It is a safe guide to follow. Unproven theories are eschewed, and the treatment advised is based on the more accepted pathological teachings. This practical treatment of the subject makes the book particularly valuable to the general practitioner. The specialist will find in the work a critical review of the most recent additions to orthopedic surgery with the views of the author as to their

value. The physiological and pathological side of orthopedics is not so fully treated as would be the case were the work from a German source, neither is there the multiplicity of devices and discussions on mechanics that one would expect from a writer on this side of the water; but there is a happy medium in the treatment of the subject which will be found satisfactory. We are somewhat surprised that apparently preference should be given to the osteoclasts of Robin and Collin over that of Grattan. The reverse is the case here. The author is evidently ingenious, as shown by his instruments for toe-drop and scoliosis, and by his operations for coxa vara, and reaching anterior spinal caries by trephining through the pedicle, lateral processes, and heads of the ribs. He advises arthrodesis at times for the hip and knee—an operation which we believe to be of rather limited value.

The views expressed on lateral curvature are interesting. We quote: "Now, no mere gymnastics of any kind, Swedish, German, American or English, will *cure* a genuine case of scoliosis. It is, however, quite otherwise with the element of mere *attitude* in cases either of genuine scoliosis or of neuromimesis." He advocates particularly the use of a poroplastic jacket with anterior supports.

A most excellent chapter is that on the application and management of plaster-of-Paris, poroplastic felt, etc.

A MANUAL OF OTOLOGY. By GORHAM BACON, A.B., M.D., Professor of Otology in Cornell University Medical College, New York; Aural Surgeon, New York Eye and Ear Infirmary. With an introductory chapter by CLARENCE J. BLAKE, M.D., Professor of Otology in Harvard University. Second edition, revised and enlarged; 114 illustrations and three plates. Pp. 422. New York and Philadelphia: Lea Brothers & Co., 1900.

IN the new edition of Bacon's excellent manual there has not been occasion to change the instruction so well imparted in the first publication (less than two years before), and it is mainly the additions that need comment here. There are changes in the type and section headings, which, with new illustrations, improve the appearance, and a colored plate is added, fairly portraying nine conditions of the drum-head. Especially notable is the extended introduction by Professor Blake, with its comprehensive view of the otological field and its reiterated demand for more intense but broad-minded specialism. Preaching only what he practises, no one can better than Dr. Blake insist upon the need of profound preparation in anatomical, physiological, and physical study for the making of a real specialist, nor can more consistently urge maintaining touch with all of the general aspects of medicine and enlisting the aid of the family physician in the study and care of aural cases. There is correction in the anatomical and other chapters of some of the minor points which the reviewer previously criticised, and new paragraphs enforce some of the lessons which before in the very condensed work seemed too briefly treated. Especially is the matter of intravenous transfusion of salt solution carefully described in detail, with the notable gains which it has made in combating shock after operations upon the sinus and cerebrum. Yet there is little change of

the simple, direct, personal treatise which appeals so strongly to most of its readers; and one has to know the huge experience and ripe skill of the author to read between the lines of this unassuming manual how much more fully every topic might have been expounded and illustrated. The presentation of illustrative cases sparingly used is particularly happy both in their choice and in the terse, clear way in which they are narrated. The work promises to hold its place at the head of the list of otological text-books for the student. B. A. R.

DISEASES OF THE GENITO-URINARY SYSTEM. A THOROUGH TREATISE ON URINARY AND SEXUAL SURGERY. BY EUGENE FULLER, M.D., Professor of Genito-urinary Diseases in the New York Post-graduate Medical School, etc. New York: The MacMillan Company, 1900.

THIS work of Dr. Fuller's occupies the rather unique position, as noted on the title page and in the preface, as being one devoted solely to the surgery of the genito-urinary tract, the consideration of venereal diseases forming no part of the volume whatever. That it should require 700 pages to discuss this subject fully only goes to demonstrate further the remarkable strides this special branch of surgery has made in the past few years. The author, in his preface, states what must be apparent to all readers of the various systems of medicine and surgery, that the main fault to be found with them lies in the fact that they have not been the product of a single mind, and that as a result the articles upon genito-urinary surgery in such works have been so often put into the hands of general surgeons that they are found to contain little more information upon the subject than could be gathered in works on general surgery. While acknowledging the correctness of this criticism, we are hardly ready to accept the plea of Dr. Fuller for a still further separation of the field of surgery into genito-urinary surgeons and specialists in venereal diseases. This proposition is one that would appear to us should be settled according to the special bent of the surgeon's mind; to make two distinct branches of medicine out of what is inseparably connected, logically and anatomically, seems to be specialism carried beyond the limit. As a treatise devoted solely to the treatment of the purely surgical problems of the genito-urinary system, the author has given us one of the very best works upon this subject before the medical public to-day. The chapter devoted to the treatment of stricture and the subject of catheterization is especially to be commended. We particularly note the writer's restrictions upon the indiscriminate irrigations of the urethra so much in vogue in certain quarters, which are known to cause great damage to the mucous membrane of the urethra when improperly or injudiciously applied. As might be expected from Dr. Fuller, considerable attention is devoted to diseased conditions of the seminal vesicles. As the author has been a pioneer in this field of genital disease, his statements upon this subject can be said to be authoritative. In discussing the matter of hypertrophy of the prostate the author gives his personal preference to prostatectomy where any radical operation is to be performed upon the gland. As regards the Bottini opera-

tion, he calls attention to the fact, which is only too apparent, that the cicatricial stenosis resulting from the cautery will gravely complicate a subsequent prostatectomy in cases where the Bottini operation has failed. A review of this most valuable contribution to genito-urinary surgery would be incomplete were we to fail to call attention to the artistic work of the publishers in giving us a volume the type of which is almost perfect, the plates chiefly new and all excellently finished.

H. M. C.

HANDATLAS DER ANATOMIE DES MENSCHEN, IN 750 THEILS FARBIGEN ABBILDUNGEN MIT TEXT. Mit Unterstützung von WILHELM HIS, Bearbeitet von WERNER SPALTEHOLZ. Dritter Band, I Abtheilung. Leipsic: S. Hirzel, 1900.

THE earlier parts of this work were reviewed at some length in this JOURNAL. The fasciculus now under consideration, Part I. of the second volume, treats of the alimentary and the respiratory systems, the urinary apparatus, and the male and female generative organs. This part includes 139 pages of text and illustrations, the latter numbering 159, many of them being colored.

The illustrations are exceedingly attractive in appearance, and, for the most part, are of great value; but they repeat the fault of very many current anatomical pictures in being too diagrammatic and in thus lacking realism. This is notably true, for example, of the representation of the submaxillary gland in Fig. 541, and of the œsophagus in Fig. 556, and of many of the pictures of the abdominal viscera.

While so much may be said in criticism, there is much to be said in praise of the work. Each of the departments of anatomy considered in this part is thoroughly illustrated, and many of the illustrations are new as well as original. The depicting and naming of the different parts of the several regions of the body is a feature of this as of the earlier portions of the work. Especially to be commended in this regard is Fig. 513, "the oral aperture and its environs."

The text is clear and concise, and, though brief, is sufficiently full to elucidate the drawings.

J. C. H.

APPENDICITIS AND ITS SURGICAL TREATMENT. By HERMAN MYNTER, M.D. Philadelphia: J. B. Lippincott Co., 1900.

IN this little book the author has presented briefly and systematically the subject of appendicitis. He has quoted the views of many authors, both at home and abroad. He has stated clearly and concisely conclusions based upon his study and practice. The book is a résumé of the literature of appendicitis, together with the personal experience of the author.

The ideas regarding appendicitis held by surgeons of different nationalities are stated very attractively. To one familiar with the subject this book is of little real value. To one looking for a brief statement of the salient facts concerning the disease, the book is somewhat attractive.

C. L. S.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Treatment of Werlhoff's Disease by the Intravenous Injection of Bichloride of Mercury.—LUSIGNOLI (*Archives Générales de Médecine*, January, 1901, p. 27), in an article on Werlhoff's disease (purpura hemorrhagica) strongly supports the view that this affection, together with scorbutus, purpura, and peliosis rheumatica, is probably due to a general bacterial infection. He points out that in Werlhoff's disease some observers think that there is a specific micro-organism and that others believe that various pyogenic organisms may be responsible for the symptoms. Thus the staphylococcus aureus and staphylococcus albus have both been found in the blood cultures. His own experience has been that the disease is not due to a specific micro-organism but to various pyogenic bacteria.

Regarding Werlhoff's disease as a bacterial infection, Lusignoli was induced by the success attending the intravenous injection of quinine in pernicious malaria, recommended by Bacelli, and also of bichloride of mercury in syphilis and in some cases of erysipelas, to give intravenous injections of bichloride of mercury in this disease also. The injections are made once daily until the condition of the patient is materially improved. They are made into the basilic, cephalic, median basilic or median cephalic vein, according as to whether one or the other happens to be the most prominent. From 1 to 4 milligrammes are given at each injection. The injections are made with an "ordinary Pravatz syringe." He cites in detail only one case of Werlhoff's disease in which this treatment was used. The patient had numerous skin ecchymoses and there had been several severe attacks of hæmatemesis. The patient recovered after several injections. No mention is made of a bacteriological examination of the blood. In another case he was apparently not so successful, for he merely states that the patient came under observation too late for the treatment to be successful. In two other

cases the results obtained were excellent. Lusignoli also reports the cure of a case of peliosis rheumatica and of a case of scorbutus by the same line of treatment.

Lusignoli concludes his article with the following statements:

1. The hemorrhagic affections (scorbutus, Werlhoff's disease, purpura) are due to special micro-organisms, the products of which penetrate into the blood.

2. The various hemorrhagic affections are of the same nature, only differing in degree.

3. Intravenous injections of bichloride of mercury cures the affections.

A justifiable criticism of Lusignoli's conclusions is that if they are based on his experience as given in his article they are drawn from insufficient data. The suggestion as to a means of treating these hemorrhagic affections is interesting, however.

Suppurative Complications of Typhoid Fever.—PROCHASKA (*Deutsch med. Wochenschrift*, 1901, xxvii., 132) examined bacteriologically twenty-two cases of suppurative complications of typhoid fever in Eichhorst's wards. For the most part the observations were made upon deep abscesses which extended into the muscular tissue, and also periosteal abscesses. In many patients the points of suppuration were multiple. In one instance a general sepsis developed. For the most part these suppurations developed late in the course of the disease. In but a few instances was there any rise of bodily temperature as a result of the complication, most of the cases appearing after defervescence. The bacteriological examination showed staphylococci in the majority of cases. Only six times were mixed infections or other organisms to be found; streptococci in pure culture in two cases; streptococci and staphylococci together in two. In one case in which otitis media followed a severe angina staphylococcus aureus and diphtheria bacilli were obtained in the pus, although the latter had not been found in the throat. In one case, that of a deep gluteal abscess, characteristic typhoid bacilli were found in pure culture.

A Contribution to the Study of the Tendon Reflexes in Typhoid Fever.—REMLINGER (*Revue de Médecine*, 10 Janvier, 1901, No. 1, p. 46) records that the first reference found in the literature to the condition of the reflexes in typhoid fever was made by Strümpell in 1878. He found that they were increased during convalescence from the disease. Several reports on the same subject have appeared since that date, and these the writer briefly reviews. The results of the investigation have been most contradictory. The patellar reflex was found diminished by Petit, Clerc, Olive, de Fleury, Delom-Sorbé, and Beaujeu. It was claimed to be exaggerated by Strümpell, Gilbert Ballet, Pluyand, Perret and Devic, and Renard. According to Albouze, the patellar reflex is diminished during the acute stage of the fever, and increased during convalescence. Renard believed that there was a close parallel relationship between the knee-jerk and the foot clonus. On the other hand, Albouze held that when the knee-jerks are exaggerated the foot clonus is diminished, and *vice versa*.

The previous results having been so variable, Remlinger decided to study the condition of the tendon reflexes in 100 cases of typhoid fever. The reflexes that he observed particularly were the patellar tendon reflex,

patellar clonus, foot clonus, and great toe clonus elicited by percussion over the tendo Achillis. He gives clinical notes of twenty cases to illustrate the variations found in the state of the reflexes. His own results show that the effect of toxins in typhoid fever is by no means a constant one. In 29 cases the reflexes were abolished; 3 of these, or 10.31 per cent., died. In 17 cases the reflexes were diminished, death occurring in 2 cases, or 11.64 per cent. In 22 cases they were normal, death occurring in 3 cases, or 9.99 per cent. In 32 cases the reflexes were exaggerated. In the text he only accounts for 28 cases in this group, and states that 7 of the 28, or 25 per cent., died. Remlinger states that whereas there does not appear to be a very definite relationship between the severity of the typhoid attack and the effect on the reflexes, yet it would appear that exaggeration of the reflexes occurs in the severest type of cases, and in the forms which he designates as ataxic and ataxo-adynamic.

Case of Hour-glass Stomach and Pyloric Stenosis; Gastro-enterostomy; Death.—This case, reported by MARTIN and POLLARD (*British Medical Journal*, December 8, 1900, p. 1635), is one of great interest. The patient was a young single woman, aged twenty-five years, who was admitted to the University College Hospital, London, complaining of pains in the stomach, vomiting, and flatulence. She had had gastric symptoms for ten years. For the first five years they had not been severe, but for the last five years they had been marked, commencing with vomiting after taking food. Severe pain preceded the vomiting, which relieved it. There had never been hæmatemesis. Vomiting, pain, and flatulence continued up to the time the patient was admitted to the hospital. For two years she had been able to take only soft articles of food.

On examination the skin showed quite marked pigmentation. The epigastrium was unduly full. Irregular peristaltic waves were observed passing from left to right. Large amounts of material were vomited. These facts indicated dilatation of the stomach, with some obstruction. The stomach contents showed the usual features of dilatation and stagnation, including yeast cells and sarcinæ on microscopical examination. There was marked hyperchlorhydria. The amount of free HCl was 0.3 per cent. The washing of the stomach showed that there was constant secretion into it. An interesting observation in connection with subsequent developments in the case was made by the house surgeon, who was in immediate charge of the patient. He found that on emptying the stomach and then introducing a known quantity of fluid into it he was unable to recover as much as he put in. The importance of this was not recognized at the time. The subsequent explanation was that the fluid had escaped from the cardiac into the pyloric compartment. No tumor could be felt, and malignant disease seemed improbable from the long duration of the symptoms. The case was regarded as probably one of pyloric constriction due to a cicatrized ulcer, although there had been no hæmatemesis.

An operation was performed on April 27, 1900. In the neighborhood of the pylorus a hard mass three or four inches in diameter was detected. It involved the pyloric end of the stomach, the first part of the duodenum, the gastrocolic omentum, and the first part of the transverse colon. Gastro-

enterostomy was considered the operation indicated. An anastomosis between the stomach and duodenum was performed. No suspicion of an hour-glass contraction of the stomach was held at the operation. The patient had frequent attacks of vomiting after the operation, and died four days later.

The autopsy showed a remarkable condition of the stomach. The viscus was markedly dilated, and was divided into two compartments by an hour-glass constriction separating the fundus from the middle and pyloric regions of the stomach. In addition there was stenosis of the pylorus. The first constriction was extreme, and was about half an inch in width and a quarter of an inch in length. On the proximal side of the constriction there was a small irregular ulcer which had perforated and formed a small abscess at the lesser curvature border. The pyloric opening was only one-quarter of an inch in diameter. It led into a markedly constricted first part of the duodenum and also into an abscess cavity two and one-half inches in diameter. The abscess was due to a perforation beyond the pylorus.

The gastro-enterostomy had been made between the second compartment and the duodenum, and consequently no relief resulted. The writers state that had the hour-glass constriction been recognized at the operation communication between the two compartments would have been established either by gastroplasty or by gastro-anastomosis with probably different results.

The authors think there is no adequate explanation for the first stricture, but think it probably due to chronic ulceration which led to persistent contraction of the circular muscular fibres. They think that the pyloric constriction was due to a duodenal ulcer.

On the Relation of Chronic Interstitial Pancreatitis to the Islands of Langerhaus and to Diabetes Mellitus.—In 1869, Langerhaus, who was the first to make a careful histological study of the pancreas, described groups of cells situated between the acini and differing markedly from those of the ordinary glandular type. Subsequently these groups of cells have come to be designated the "islands of Langerhaus." Some have held that these islands consisted of follicles of lymphatic tissue; others that they were formed during embryological life and subserved some special function. Lewaschew believed he could multiply them by prolonged stimulation, by overfeeding, or by the repeated administration of pilocarpine. Laguesse, Schäfer, and Diamare suggested that they furnish an internal secretion which influences carbohydrate metabolism. The only evidence in favor of this view is that quite recently advanced by Ssobolew, who found that the cells of the islands became more granular after feeding animals on carbohydrates. He observed that the islands of Langerhaus were not involved in the sclerotic process which ensues after the ligation of the duct of Wirsung in dogs. He believed that this fact explains the absence of glycosuria after ligation of the duct.

Opie (*The Journal of Experimental Medicine*, vol. v., No. 4, January 15, 1901) has made a special study of these islands in man and in lower animals, in infected specimens, and in glands stimulated by the administration of pilocarpine, and finds: 1. That the islands of Langerhaus are composed of cells having the same origin as those of the glandular acini, but forming

structures which are independent of the secreting apparatus and in intimate relation with the vascular system. 2. That in the splenic end of the cat's pancreas they have a definite position within the lobule, each of which contains one of these structures. 3. That in the human pancreas they are more numerous in the splenic extremity or tail than elsewhere. 4. That prolonged stimulation of the gland does not, as claimed by Lewaschew, transform groups of acini into the islands of Langerhaus.

Opie has studied the relation of chronic interstitial pancreatitis to the islands of Langerhaus both in infants and adults. He states that there has never been a satisfactory classification of cases of chronic interstitial pancreatitis in adults. From a study of the cases which have come under his observation he recognizes two distinct types: 1. The interlobular, in which the connective tissue is increased between the lobules, the intralobular or interacinar tissue being little, if at all, increased. 2. The interacinar, where the interlobular tissue is only slightly altered, while fibrous tissue, which replaces the parenchyma, separates individual acini. The subject is discussed at considerable length, and the histological findings in several cases is given. There is also an interesting consideration of the relation of interstitial pancreatitis and the lesions of the islands of Langerhaus to diabetes mellitus.

Opie sums up the results of his studies as follows:

1. Congenital syphilitic pancreatitis retards the development of the glandular acini but does not affect the islands of Langerhaus. Embedded in the stroma, but not invaded by it, the latter maintain their continuity with the small ducts and acini with which they have a common origin.

2. Two types of chronic interstitial inflammation affecting the developed pancreas are distinguishable:

(*a*) Interlobular pancreatitis. In the interlobular variety the inflammatory process is localized chiefly at the periphery of the lobule and implicates the islands of Langerhaus only when the sclerotic process has reached a very advanced grade. When pancreatitis has followed obstruction of the ducts the islands long remain unaltered, though embedded in dense scar-like tissue.

(*b*) Interacinar pancreatitis. In the interacinar type the process is diffuse, invading the lobules and separating individual acini. The inflammatory change invades the islands of Langerhaus.

3. A relationship has been observed between lesions of the islands of Langerhaus and the occurrence of diabetes mellitus.

(*a*) In one of eleven cases of interlobular pancreatitis diabetes of mild intensity occurred. The sclerosis, which in this case followed obstruction of the ducts by calculi, was far advanced and affected the islands of Langerhaus.

(*b*) In two of three cases of interacinar pancreatitis diabetes was present. The third case was associated with a condition, hæmochromatosis, which at a later stage is associated with diabetes, the result of pancreatic lesion.

(*c*) In a fourth case of diabetes hyaline deposit between the capillaries and the parenchymatous cells had so completely altered the islands of Langerhaus that they were no longer recognizable.

Opie is strongly of the opinion that lesions of the islands of Langerhaus are very intimately associated with the etiology of the cases of pancreatic diabetes.

A Report on the Cases of Typhoid Fever Admitted into the Royal Victoria Hospital (Montreal) During 1900.—GILLIES (*The Montreal Medical Journal*, February, 1901, p. 99) reports that 151 cases of typhoid fever were treated in the Royal Victoria Hospital in 1900. Of these, 76 were males and 75 females. As 22 of the cases were still under treatment at the time the report was issued, the statistics are made up from the balance of the cases, or 129 in all. Of the 129 cases, 10 died, giving a mortality of 7.7 per cent.

The third decade claimed the largest number of cases. There were 55 patients between the ages of twenty and thirty years. Regarding the symptoms, it is interesting to note that diarrhœa was present at the onset in 25.6 per cent. of the cases. Vomiting occurred at the onset in 26.3 per cent., and during the course in 13.9 per cent. of the cases. Rose-spots were present in 69 per cent. of the cases, and there was a palpable spleen in 61.2 per cent. Relapses occurred in 5.4 per cent., the shortest and longest duration of the relapse being eleven and twenty-two days respectively. In one case the interval between the initial attack and the relapse was three months. The Widal reaction was reported on in all but 4 cases. In only 1 case was it negative throughout the course of the disease. The earliest day on which it appeared was the fourth, and the latest the thirty-third day. Ehrlich's reaction was present in 34 per cent. of the cases.

Meteorism was present in 20 per cent. Perforation of the bowel occurred in 4 cases. Of these 4 cases 1 recovered after operation. The operation was performed eighteen hours after the time the perforation occurred, which happened on the twelfth day. Intestinal hemorrhage occurred in 12 cases, and was the direct cause of death in 3 cases. Femoral phlebitis occurred in 7 cases, and brachial phlebitis in 1 case. Suppurative otitis media occurred in 4 cases, and periostitis in 3 cases.

A Case of Myositis Ossificans Progressiva.—WILKINSON (*The Quarterly Medical Journal*, November, 1900, p. 24) reports a case of myositis ossificans progressiva in a boy, aged thirteen years. The boy was healthy until he was two and a half years of age. About this time some isolated hard lumps were observed on his back. These gradually extended and joined together. When the boy came under observation at the age of thirteen, a striking bony mass extended over the back from immediately to the right of the lowest lumbar spine, passing over to the left in the lower dorsal region, and thence spreading over the left scapular and interscapular regions, and crossing the upper dorsal spines to the inner border of the right scapula at its upper part, the whole mass much resembling a single branched antler of a stag. There were other nodules over the back. The bony masses were fixed to the spine and ribs beneath, and the skin was only slightly movable over their more prominent parts. The whole back was rendered stiff, and he had never been able to entirely dress himself on this account. The boy was unable to open his mouth owing to supposed involvement of the temporal muscles. In-

creased firmness of or definite nodules in various other muscles were made out. Thus, certain of the neck, arm, anterior thoracic, abdominal, thigh, and leg muscles were involved. The thumbs and great toes presented the condition known as microdactyly. Wilkinson noted that some of the muscle nodules had disappeared since the patient came under his observation. As a rule, the nodules had developed without pain being complained of.

Lorenz, in Nothnagel's *Specielle Pathologie und Therapie*, had collected statistics of 51 cases of this rare disease. Of these, 32 were males, 13 females, and in 6 the sex was not stated. Little is known concerning the pathology of the disease. The name myositis is held to be misleading. The deposit commences in the fibrous tissues, and the muscle fibres are never affected except by secondary atrophy. The deposit at first consists of small round cells and fibrous tissue. Sometimes cartilage is found in this, which later undergoes ossification, but more generally bone is found without the occurrence of cartilage (Zeigler). Wilkinson states that the most generally accepted view of the pathology of the disease is that advanced by Pincus, who believes it to be a new-growth and compares it to a multiple fibroma. Treatment is without avail. Death usually results from increasing fixation of the thorax resulting in suffocation, or from some intercurrent pulmonary disorder.

SURGERY.

UNDER THE CHARGE OF

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The Treatment of Appendicitis.—VERNEUIL (*Jour. de Chir. et Annales de la Soc. Belge. de Chir.*, January and February, 1901) states that, as has been observed by Broca, the true question in those cases of acute appendicitis with a circumscribed peritonitis is: Is a non-operative treatment capable of aborting a crisis if it is instituted in time, and is it to our interest whenever possible to secure this abortion and in place of operating immediately to perform an operation at some later date? To this question the author responds in the affirmative. The general opinion is that it is best to perform appendectomy if the appendix presents itself in the field of operation; but, on the other hand, it is well to guard against an extensive search and all manœuvres that might have the effect of breaking up adhesions. In conclusion it may be said: 1. That if the symptoms at the onset of the disease are very grave (vomiting, first of the stomach contents, then bilious, and, finally, fecal; constipation with no passage of gas; pain in the iliac

fossa and at McBurney's point; marked tympany; dyspnœa; either a marked elevation of temperature or one that is subnormal; a pulse of bad quality, and a shrivelled face), or if there be the symptoms of a general purulent peritonitis or a diffused septic peritonitis, one should operate immediately; performing a median cœliotomy followed by appendectomy and drainage. 2. If the symptoms of the onset are those of acute appendicitis with circumscribed peritonitis (sharp pain at McBurney's point, vomiting, constipation, more or less temperature with a small regular pulse in proportion to the temperature, moderate tympany, face less shrunken and general condition fairly good); if some time has passed since the onset and one finds in the right iliac fossæ muscular rigidity and the symptoms that denote the presence of a pseudomembranous exudate, with inflamed and adherent omentum, one should adopt medical treatment (ice to the abdomen, opium, absolute rest in the dorsal position, and iced Vichy water in small quantities). The patient should be carefully watched, and the surgeon ready to intervene (*a*) if the symptoms become aggravated and if there be evidence of an extension of the peritonitis; (*b*) if the inflammatory tumor becomes larger; (*c*) if the temperature persists after forty-eight hours the tympany remaining stationary; (*d*) if the tympany shows no tendency to disappear or remains about the same. It is necessary when one operates to confine one's work to the periappendicular space; no attempt should be made to remove the appendix unless it presents itself and its removal can be accomplished without much difficulty. If this cannot be done drainage should be introduced and the appendix left *in situ*, to come away later as a slough. The best incision in these cases is that of Roux, in which the periperitoneal incision is on a line with the more deeply seated pus. 3. After repeated attacks of appendicular colic or of acute appendicitis without peritonitis, and in all of those cases after a second attack, one should perform appendectomy. The incision of choice in these cases is that of Jalaguier.

The Sterilization of the Silk Catheter.—HERMAN (*Centralblatt für Chir.*, January 19, 1901) states that a safe and convenient method of sterilization which will be suitable to all kinds of catheters is still to be discovered. The metal and the Nélaton (French) catheters are best sterilized by boiling. This method, unfortunately, cannot be used with silk catheters, which become rough and cracked if treated in this manner. In an effort to find a solution in which silk catheters could be boiled without injury, a saturated solution of ammonium sulphuricum was tried with the following results: 1. Silk catheters will stand several hours' uninterrupted boiling in this solution without apparent injury. After five hours of consecutive boiling they retained on their outer and inner surfaces a shiny appearance, did not become stiff, and, in fact, they became more elastic than ever. 2. They were found to be uninjured after repeated use and boiling at intervals. 3. Old catheters that are infected may be easily sterilized by boiling them for three to five minutes in the ammonium sulphate solution. 4. The catheters may be used as soon as they are removed from the solution. It is not necessary to dry them or remove any of the solution that may be adherent, as in not a single instance has irritation or inflammation of the urethra followed their use even when used immediately from the solution. 5. This solution is

equally adapted for the sterilization (by boiling) of Nélaton catheters, and all other kinds of urethral instruments. At the present time there is no other method of sterilization of the silk catheter that combines such simplicity of technique and such absolutely good results.

Total Suture of the Bladder after Suprapubic Cystotomy in the Presence of Old Infected Calculi.—LOUMEAU (*Annales de la Policlinique de Bordeaux*, February, 1901) states that in the case of the bladder, as in all other operations, suturing is the method of choice, while drainage should be considered as the method of exception and of necessity. The author reports the case of a man, aged eighty years, who presented himself with a bad cystitis and the symptoms of a large vesical calculus. Operation being decided upon, suprapubic cystotomy was performed in the usual manner, and three large phosphatic calculi removed. The bladder was then closed by a double row of catgut sutures, and the operation completed by the insertion of two small pieces of gauze to drain the prevesical space. The patient made an uninterrupted recovery, with the exception of a slight attack of phlebitis of the left leg, which soon responded to appropriate medical treatment. Three weeks after the operation the patient returned to his home, the phlebitis had entirely disappeared, and in every way there was a marked improvement in the patient's general condition. Immediate suture of the bladder markedly diminishes the period of convalescence, and is in all cases an operation that may be performed without danger.

Tuberculosis of the Vesiculæ Seminales, Testis, and Prostate; Complete Excision of the Right Side; Incision and Curetting on the Left Side; Cured.—WALKER (*Maryland Medical Journal*, February, 1901) reports the case of a man, aged twenty-seven years, of non-tubercular family history, who presented himself for treatment with a tubercular infection of the genitalia of three years' duration. Examination showed a sinus leading down through the scrotum to the diseased right testis, which had undergone atrophy and had entirely lost the characteristics of the normal gland. The cord was uninvolved, but there was marked inflammation of all the tissues in the lower right side of the scrotum. Rectal examination disclosed on the right side an induration of about one-half of the seminal vesicle, and the same hardening in the ejaculatory ducts. The induration extended to and apparently entered the prostatic substance. On the other side the involvement was not so marked, but there was a distinctly hard infiltration, about midway of the seminal vesicle, which extended toward its apex. The ejaculatory duct on this side was soft and uninvolved. The prostate was not enlarged though slightly nodular, but everywhere soft, and presented no suspicion of disease, except where the right ejaculatory duct entered. Endoscopic examination of the bladder and urethra showed them to be normal except for a small hemorrhagic area in the posterior portion of the urethra, four centimetres in front of the prostate, probably the remains of a specific urethritis contracted five years before the onset of the tuberculosis. The urine was negative for tubercle bacilli, except on one occasion, when three very suspicious looking bacilli were found. The operation consisted in an incision in the right inguinal region, the removal of the right testis, and all

the adherent diseased tissue. The incision was then carried upward and the abdomen opened. The cord was followed down and the seminal vesicles exposed. As it was found almost impossible to completely expose the seminal vesicles without separating the bladder from the surrounding structures, the patient was placed in the Trendelenburg position, and the perineum opened in the median line. The cord was pushed through this opening and the vesicles then came well into view. The right vesicle was then excised along with about one-third of the prostate gland. The left testis was not removed; the left seminal vesicle was incised, then thoroughly curetted, touched with pure carbolic acid, and finally packed with iodoform gauze. The left testicle was incised and found to be the seat of a moderate sized abscess, which was curetted, touched with pure carbolic acid, and finally packed with iodoform gauze. The epididymis was practically obliterated by the disease, and markedly infiltrated. The cord was normal, and there were no adhesions to the tunica. The abdominal wound was closed with drainage, the perineal wound was packed with gauze, and a catheter introduced into the bladder through the urethra. The patient made practically an uninterrupted recovery, and when examined, six months later, the following condition was found: The perineal wound and the wound on the right side of the scrotum had entirely healed, the left testicle was soft, and in no place indurated or hard; free in the scrotum except over upper part of the incision; nearly normal size; not tender nor painful. Examination of the rectum revealed an entire disappearance of the disease, which was present on the left side. The tissues show absolutely no evidence of disease. The patient's general condition has undergone marked improvement, and he has gained twenty pounds in weight. The urinalysis is entirely negative, and micturition is perfectly normal. The author emphasizes the importance of the combination of the inguinal and perineal incisions in the cases where complete excision is attempted. These incisions allow very much better working room, permit a more careful dissection, bring the parts better into view, and last and most important of all it permits of perineal drainage, which is the direction in which the discharges naturally tend to go. In the inguinal incision alone the bladder must be more extensively separated from the surrounding tissues and a very much larger wound is the result. This, of course, necessitates leaving large and deep pockets, which are practically almost impossible to drain. In conclusion, it may be said that this case adds some evidence in favor of conservative surgery.

Analgesia from Spinal Subarachnoidean Cocainization.—MURPHY (*Journal of the American Medical Association*, February 9, 1901) states that since the announcement of the first death from chloroform anæsthesia the medical profession has been constantly looking for an anæsthetic that might be administered without danger. Subarachnoidean spinal anæsthesia was discovered by Corning in 1884, who suggested this as a method of treating diseases of the spinal cord, but the surgical application was not considered by him. Bier demonstrated, in 1898, that analgesia of the extremities for surgical purposes could be produced by this means. The physiological effect of the application of cocaine to the spinal cord is produced by the direct application of the cocaine to the posterior roots and ganglia, and not

to the cord itself. It is not a sensory tract paralysis, because the sense of contact is not affected, the reflexes are but slightly altered, being usually diminished, muscular sense and co-ordination are often affected, intestinal peristalsis and uterine contractions are rather stimulated than depressed, while the actions of the sphincteric muscles of the bladder, vagina, and rectum are often completely abolished. The above leads to the speculation, Cannot reflex ilius be treated by this means? The dose to be injected ranges from 7 to 20 minims of a 2 per cent. solution of cocaine in the adult, depending upon the individual peculiarities of the patient, the location and probable duration of the operation, etc. A 1 per cent. solution should be used in children. The cocaine solution should be freshly prepared at the time of the operation. The injection should be made with an ordinary sterilized hypodermic syringe.

The skin is prepared in the usual manner, and the space between the fourth and fifth lumbar vertebræ, one-half inch to the side of the median line, is the point of election. The fluid should be injected slowly, from forty to sixty seconds being consumed in the procedure. The solution should never be injected except when the cerebro-spinal fluid is flowing from the needle. The symptoms produced by the injection are, first, a sense of heat passing over the entire body, then thirst, and then in from seven to fifteen minutes nausea, and one or two attempts at vomiting occur. The former may continue for ten minutes or so with several efforts at emesis. Vomiting is preceded by all the symptoms which ordinarily accompany nausea from any reflex cause. The pulse may become scarcely perceptible at the wrist, and the facial expression is often that of profound depression, the capillary circulation, as a rule, remains good, and respiration is not materially affected. The sphincter ani is frequently relaxed, as is also occasionally the vesical sphincter. The relaxation of the vagina and the rectum, which is so common in cocaine analgesia, would theoretically be of advantage in obstetric cases. The analgesia usually appears in from three to ten minutes after injection, although it may be delayed to twenty or even thirty minutes. It usually commences at the feet and extends upward and may be complete, partial, or entirely absent. Its duration is from twelve minutes to three hours or more. The post-operative symptoms are usually headache, sometimes very severe and lasting for several days, but the rule is that it subsides in from twelve to twenty-four hours. Vertigo and ataxia are occasionally present, and may persist for five or six days; the pulse soon returns to normal, and temperature usually occurs in the evening following the injection. It is often preceded by a chilly sensation, and it is probably due to irritation of the thermic centre in the cervical portion of the cord. The patient's general condition on the following day, even after major operations, has been uniformly better than when ether or chloroform was used, and the tissue reaction to the traumatism has been less marked. Failure to obtain analgesia is often explained by faulty technique or selection of cases. It may be due to irregularity in the action of the drug in different cases and at different times in the same case, or to the use of a too small quantity of the solution. Chronic alcoholics seem to be unfavorably affected by the drug. The idiosyncrasy to cocaine is more frequent than to any other drug, and explains the absence of analgesia in many cases. Eucaine does not give such satisfactory

results as cocaine, but its use is usually followed by the same unpleasant symptoms as when cocaine is employed. Gumprecht's statistics of seventeen deaths from lumbar puncture, performed for diagnostic purposes in the presence of severe cerebro-spinal lesion have no value as a guide as to the practicability of cocaine analgesia.

Perigastric Adhesions.—BIRD (*Inter-Colonial Medical Journal of Australasia*, December 20, 1900) states that this is a very complex subject—complex in its anatomy, in its causation, and in its results—and it is only recently that its importance has been appreciated and its possibility partly understood. It is now evident that perigastric and neighboring adhesions merit especial consideration quite apart from the disease which gave them birth. Peritoneal adhesions are very common, and perigastric adhesions are said to occur in 5 per cent. of all autopsies. They vary widely in density, texture, size, and shape, are due to a variety of causes, of which syphilis is undoubtedly the origin in some cases. They tend to disappear in some instances, and in some cases may produce no symptoms, while in others they give rise to very grave ones. The author especially draws attention to the slight forms of perigastric adhesion, which may be classified by their causation thus: 1. Those induced by gastric duodenal ulcers. 2. Those whose infection arises from the bile ducts. 3. Those due to syphilis. 4. Those of uncertain origin. 5. Some cases are undoubtedly traumatic in origin. By their symptoms they may be arranged into those which cause pain as the clinical symptoms and those which constrict natural passages, such as the bile duct or the duodenum, causing jaundice and distention of the gall-bladder in the first case, and dilatation of the stomach as the chief clinical phenomenon in the other. The most characteristic symptoms of the slight perigastric adhesion is epigastric pain started or increased by any sort of food. The pain caused by an adhesion is definitely localized, the patient putting the finger right over the spot where operation subsequently proves the lesion to be, while, on the other hand, the pain of acute inflammation is often referred to a point far distant from the actual seat of inflammation. There is usually more or less pain after eating, coming on half an hour after food is taken, and so is intermediate between the immediate pain of ulcer and the delayed pain of atonic dyspepsia. Sometimes this pain is at once relieved by the patient assuming the recumbent posture. Movement either arouses or aggravates the pain. The severity of the pain is no index to the size of the adhesion, and the pain may range from a dull one to really extreme agony. In no case is the pain accompanied by an elevation of temperature. The general health may suffer much in some cases, while in others it is but slightly affected. Pain distinctly epigastric in position and bearing more or less definite relation to gastric locomotion is then the earliest symptom, and herein lies the main distinction from malignant diseases of the stomach, of which pain is not at all a marked symptom until late in the disease, when in most cases a palpable tumor has formed. Vomiting is not a prominent symptom, and when it does occur the vomited material does not show signs of gastric digestion having been delayed. The presence of jaundice is usually an accident, but in some cases it may help in forming a probable diagnosis in these cases. The futility of medicinal and dietetic treatment is

of great value, not only as a means of diagnosis, but also as an indication of the necessity of operative interference. Persistent local pain, disability of the patient, loss of general health, strength, and vigor, or the inefficiency of the physician's treatment, all point toward an exploratory laparotomy, which should be performed in the usual manner, and which is the only method which will permanently cure perigastric adhesions. Under proper aseptic precautions it is an operation that may well be regarded as being without any mortality.

PEDIATRICS.

UNDER THE CHARGE OF

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Observations Upon Fœtal Rickets.—FEDE and FINIZIO (*Revue mensuelle des Maladies de l'Enfance*, March, 1901, p. 101) continue the observations upon this condition inaugurated by Fede and Cacace and reported by them to the Congrès de Pédiatrie held in Turin in 1898 (*Pediatria*, February, 1900). [See abstract in this department of the AMERICAN JOURNAL OF THE MEDICAL SCIENCES, September, 1900, p. 353.] In this earlier communication Fede and his assistant reported the results of examinations of 500 new-born infants, among which only one case was found that could be said to have the clinical signs of rachitis, and only four with craniotabes alone. The present paper gives the results of the study of 475 infants observed in the same hospital (Maternita degli Incurabili) in Naples. Of the entire number only 3 showed some of the clinical signs of rachitis, 3 others had only craniotabes, and 4 double genu varum.

In all their observations they have frequently encountered irregularities of the cranial bones, a large anterior fontanelle, with unclosed posterior and lateral fontanelles, and open sutures. This they consider rather as an instance of insufficient development than an expression of rachitis. Microscopical examination of the cranial bones in cases presenting widely open fontanelles and separated sutures has failed to show any lesions recalling those of rachitis. None of them showed the rich vascular network which, according to Kassowitz, is never wanting even in the slightest degrees of rachitism.

The result of this second series of observations serves to confirm the conclusion reached in the earlier work, that fœtal rickets is extremely rare, and to prove in addition that in the new-born large fontanelles and separated sutures are not always indications of rachitis, but that in the majority of cases at least they prove only a retarded ossification.

The Causation of Infantile Scurvy.—C. C. CORLETTE (*British Medical Association Meeting*, August 13, 1900; *Pediatrics*, February 15, 1901, p. 142) advances the hypothesis that infantile scurvy is due to a deficiency of citric acid, which is a normal constituent of fresh milk, a quart of cow's milk containing as much citric acid as a large lemon. This acid exists in milk as a calcium salt, probably the normal calcium citrate, and may be present either in crystalline or amorphous form. The amorphous salt is more soluble in both cold and hot water than the crystalline, and by boiling for a sufficient time it is converted into the latter. When milk is heated to the boiling-point its power of holding citric acid as calcium citrate is greatly diminished, and by so much the more as the citrate is converted into the crystallizable form; and in proportion to the degree of saturation existing in the fresh milk, so is the tendency of the salt to precipitate out and be more or less lost. Since the sterilization of milk involves boiling, it is easily seen how the restriction of diet to sterilized milk may cause a deficiency in the supply of citric acid to an infant, and if deficiency of citric acid be a cause of scurvy the incidence of this disease during the prolonged administration of a cooked or sterilized milk diet is easily explained.

This hypothesis would also explain why the disease does not occur when the milk is only pasteurized, especially when the process of pasteurization is carried out on a large scale.

The following precautions are suggested for the purpose of diminishing as far as possible the loss of citric acid in the boiling of milk: Any water to be used in the formula should be added to the milk before the boiling, so as to get a less concentrated solution of the citrate than obtains in the undiluted milk, and therefore one less likely to suffer loss of this salt by its comparative insolubility after boiling. Further, the milk should not be boiled a moment longer than the occasion requires, should be allowed to cool in the vessel in which it has been boiled, and should be well stirred when cool enough, in order to redissolve the converted crystallized citrate as far as possible.

Finally, to make the milk retain better the precipitated crystals in suspension, the addition of a gummy substance before boiling is suggested. The milk, too, may possibly be made a better solvent of calcium citrate by the addition of an alkaline phosphate, such as sodium or potassium phosphate. Milk normally contains a small amount of potassium phosphate, and this salt may be one of the factors, the author thinks, in helping to make milk a better solvent of calcium citrate than is water. The addition of a small amount of an alkaline phosphate, about ten to fifteen grains to the pint, should therefore help to increase the power of dissolving the citrate.

Position of the Cæcum and the Appendix in the Child.—VALLÉE (*Thèse de Paris*, 1900) bases his thesis upon post-mortem examinations of 100 children, and draws the following conclusions:

1. The cæcum in the child occupies a higher position than in the adult. In 72 cases out of the 100 it is situated above the right antero-superior iliac spine, while in the adult it is below this point. It may be entirely to the left of the median line without inversion of the abdominal organs, as occurred in one of his cases.

2. The position of the appendix depends upon that of the cæcum, and is therefore high. In 78 cases it is entirely above a plane passing through the antero-superior iliac spines. Its mean length is about 5 centimetres, and its direction is most frequently vertical, upward or downward.

3. The colon is kept in place by a peritoneal fold which is reflected upon the posterior abdominal wall at the level of the iliac crest. This mesocolon may be wanting, and the posterior surface of the colon may be without serous envelope. The mesoappendix comes from the mesentery sometimes, but rarely from the colic peritoneum. In half the cases a peritoneal fold arises from the mesentery or from the base of the appendix to be reflected upon the iliac vessels. Other folds of peritoneum come from the appendix to form the ileocæcal fossa. In one case a serous ligament united the base of the appendix to the summit of the bladder.

Whey-cream Modifications in Infant Feeding.—FRANKLIN W. WHITE and MAYNARD LADD (*Philadelphia Medical Journal*, February 2, 1901, p. 218) present a valuable study of milk modifications with special reference to the differential modification of the proteids. Their results are briefly summarized as follows:

1. By the use of whey as a diluent of creams of various strengths we are able to modify cow's milk so that its proportions of caseinogen and whey proteids will closely correspond to the proportions present in human milk. We, therefore, render it much more digestible and suitable for infant feeding.

2. The best temperature for destroying the rennet enzyme in whey is 65.5° C. Whey or whey mixtures should not be heated above 69.3° C. in order to avoid the coagulation of the whey proteids. The percentage of whey proteids in the whey obtained by us was 1 per cent., while in the analysis of the whole milk approximately three-quarters of the total proteid was caseinogen and one-quarter was whey proteids.

3. On the basis of these analyses we were able to obtain whey-cream mixtures, with a maximum of 0.90 per cent. and a minimum of 0.25 per cent. of whey proteids in combination with percentages of caseinogen varying from 0.25 per cent. to 1 per cent.; of fats, from 1 to 4 per cent.; of milk sugar, from 4 to 7 per cent.

4. The emulsions of fat in whey, barley-water, gravity cream, and centrifugal cream mixtures were the same, both in their macroscopical and microscopical appearances. The combination of heat and transportation, such as sometimes occurs in hot weather, partially destroys the emulsion in all forms of modified milk, but this disturbance can be prevented by the simple precaution of keeping the milk cool during delivery.

5. Whey-cream mixtures yield a much finer, less bulky, and more digestible coagulum than plain, modified mixtures with the same total proteids; the coagulum is equalled in fineness only by that of barley-water mixtures. The coagulum yielded by gravity cream mixtures and centrifugal cream mixtures is the same in character.

The Glycolytic Power of the Tissues of the Nursling in Health and in Gastro-enteritis.—TERRIEN (*Revue mensuelle des Maladies de l' Enfance*, January, 1901, p. 31) states that the power of transforming sugar, as is well

known, is not confined to the liver, but is possessed in a certain measure by the other tissues of the body, and that consequently an experimental alimentary glycosuria indicates only a general glycolytic insufficiency (in liver and tissues).

In every experiment having for its object the measure of the functional power of the liver, it is therefore important, as Achard and Castaigne have shown, to diminish previously the glycogenic power of the tissues. This point, so important in investigations upon the adult, is none the less so with the nursling, and the author therefore has made some investigations to determine whether the glycogenic power of the tissues varies in notable degree with infants in good health and those suffering from gastro-enteritis. By means of subcutaneous injections of pure sterilized glucose a determinate quantity of sugar may be introduced into the general circulation, and thus by gradual increase of the dose until glycosuria appears one may readily determine with exactness the glycolytic power of the tissues in both classes of infants.

The result of these experiments shows that the glycolytic power of the tissues of the nursling is relatively high, since the quantity of sugar so convertible varied between 50 and 80 centigrammes per kilogramme of weight, while the liver arrests only about 4 or 5 grammes; that inversely with what is observed in the liver the glycolytic power of the tissues remains about constant, whether the experiment be made upon a healthy infant or upon one suffering from gastro-enteritis; that, consequently, it is not necessary to take into account this possible source of error in investigating alimentary glycosuria in the nursling.

THERAPEUTICS.

UNDER THE CHARGE OF

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Treatment of Pancreatitis.—DR. MAYO ROBSON says that operative treatment of acute pancreatitis, especially when of the suppurative variety, is alone indicated. The symptoms being so vague, however, such a radical procedure may not always be justified. Morphine for pain and aperients to relieve distention are advisable. Subacute pancreatitis is more amenable to treatment. Distention, when present, calls for gastric lavage, and turpentine enemata or calomel by the mouth. Calomel is distinctly valuable, act-

ing as it does as an intestinal antiseptic. It should be given in five-grain doses followed by a saline aperient. Diarrhœa, which is apt to supervene after the constipation is relieved, should be controlled by salol and bismuth. Abdominal section and drainage may be indicated. Chronic pancreatitis should be treated by abdominal section and drainage.—*British Medical Journal*, 1901, No. 2106, p. 1129.

Treatment of Strychnine-poisoning by Inhalations of Oxygen.—Dr. M. C. Osterwald draws attention to the early studies of von Leube and Rosenthal, who showed the widest beneficial results of artificial respiration in strychnine-poisoning, convulsions ceasing or markedly diminishing while the respiratory movements were being practised. Two theories have heretofore been in vogue to explain this phenomenon. According to one the increased respiratory movements bring about accelerated gaseous exchange, which in its turn favors the organic combustion of the poison circulating in the blood. Another hypothesis attributes the results to the reflex action on the pneumogastric. In order to determine the correct position if possible, the author substituted oxygen inhalations for artificial respirations. Early experiments convinced him that it was not possible to accustom animals to the action of strychnine, the same dose producing similar effects in every trial. This afforded a definite starting-point. The influence of free oxygen was very marked. Guinea-pigs would take more than double the convulsive dose if breathing oxygen, and, on the contrary, they succumbed very rapidly to a much smaller dose if the air was below its normal oxygen content. The results indicate a practical procedure in the treatment of this fortunately rare form of poisoning.—*Archiv für experimentelle Pathologie und Pharmakologie*, 1901, vol. xliv., S. 451.

Interesting Case of Idiosyncrasy.—Dr. Sutherland details the aberrant effects of quinine, morphine, and strychnine in a woman, aged thirty-three years, who has been in good health at least since 1887. Quinine even in small doses produces a universal nettle-like eruption. The same phenomenon is noted after bathing in salt water and also in warm weather, thus showing a heightened skin irritability. Morphine acts only as an irritant. Thirty minutes after taking a dose she is nervous and restless and has a sensation of impending death. Strychnine causes a marked itching of the palms of the hands and soles of the feet. This sensation of itching becomes universal if the dose is increased. Suggestion plays no part in the phenomena.—*Münchener medicinische Wochenschrift*, 1901, vol. xlviii., S. 525.

Sodium Persulphate and Metavanadate.—Dr. Albert Robin in a short note comments on the use of sodium persulphate and metavanadate in tuberculosis. He has found that these remedies have no special action, but that both stimulate the digestion very markedly, and their beneficial action is thus to be explained. Of the persulphate of soda he prescribes: Sodium persulphate, 2; water, 300. A tablespoonful should be administered one-half hour before meals. If this remedy does not stimulate the appetite within six days it is useless to continue its administration. As far as the metavanadate is concerned, some patients are acted on better by it; some by the per-

sulphate. In general he has found that the metavanadate has a better action. It is prescribed: Sodium metavanadate, 1; water, 5000. This is given in teaspoonful doses half an hour before meals. The remedy should not be continued for more than four days consecutively.—*Les Nouvelles Remedies*, 1900, vol. xvii. p. 49.

Antagonism of Atropine and Morphine.—Dr. Ernst F. Bashford has contributed an excellent pharmacological study bearing on the mutual antagonism of these drugs. One point at least of therapeutic interest is brought out, and that is that the antagonism is said to be not as pronounced as others have claimed, notably Binz and Kobert. The former in cases of opium-poisoning administers as much as one-sixth to one-tenth of a grain of atropine, while Kobert recommends one-sixtieth of a grain every half hour. Bashford claims that these doses are far too great, and recommends that a single dose only of one forty-fifth of a grain should be given.—*Archives Internationales de Pharmacodynamie et de Therapie*, 1901, vol. viii. p. 311.

Urotropin in Pyelitis.—Dr. J. Pollock Simpson reports on the use of this drug in acute and chronic cystitis. In one case reported the urine was markedly fetid and much pain was experienced during urination and in the interval. Six-grain doses relieved the pain and cleared up the urine. The odor disappeared and the pus quickly disappeared, and in a few weeks the patient was free from severe paroxysmal attacks of cystic colic. In another case a chronic cystitis was accompanied by an ulceration at the base of the neck of the bladder. Notwithstanding free curetting of the ulcerated area, improvement did not follow until after the administration of this remedy.—*The Therapist*, 1901, vol. vii. p. 81.

Dryopteris Spinulosum.—Dr. H. Wolff reports the use of the ethereal extract derived from this substance in place of that of male fern in six cases of tænia in children aged from ten months to thirteen years. The action was satisfactory, and no untoward effects were observed.—*Archiv f. Kinderheilkunde*, 1901, vol. xxxi., S. 74.

[The experience of others working with *dryopteris spinulosum* is in accord with these observations.—R. W. W.]

Typhoid in Children.—Dr. H. Stowell says that intestinal antisepsis is of prime importance. Laxatives are called for during the first few days. Calomel, one-tenth of a grain every hour, is antiseptic to some degree, and promotes glandular activity in the liver and intestines. Salol is his favorite drug. This is an excellent antifermentative and antiseptic. It breaks up in the intestine and becomes carbolic acid and salicylic acid, but untoward results are not common. Magnesium citrate during the first week is advised. Good results from using the well-known tablets of podophyllin, calomel, guaiacol, and menthol are reported. During the third week the soft, compressible pulse of a flagging heart needs help. Strychnine, digitalis, camphor, and ammonia will spur it on by increasing the systole. Coffee may be given by mouth or per rectum. Occasional doses of antipyretics of the

coal-tar group may be given in particular cases and be very helpful, but their effect on the circulation should be watched.—*Archives of Pediatrics*, 1901, No. 4, p. 271.

Anti-typhoid Inoculations.—Dr. A. E. Wright reports on the results obtained by such inoculations in Egypt and Cyprus during the year 1900. Of a total number of 2669 uninoculated and 720 inoculated soldiers, 68 of the former and 1 of the latter contracted the disease. The proportion, therefore, was 2.50 to 0.14 of incidence. The reduction was nineteenfold. The one patient of the inoculated series was taken ill shortly after the time of inoculation, and it is possible that he had the disease prior to this.—*British Medical Journal*, 1901, No. 2105, p. 1072.

Treatment of Rheumatoid Arthritis.—Dr. P. W. Latham, basing his treatment on the hypothesis that rheumatoid arthritis is a disease of the trophic centres situated in the spinal cord, advocates the use of continuous counter-irritation along the spine in the early history of the acute poly-articular variety. When the disease is advanced, however, the ends of the bones enlarged, and the cartilage destroyed, one cannot hope to reduce such by counter-irritation. In these chronic cases, however, in which there are acute exacerbations of pain and swelling in some of the larger joints, the application of blisters to the spine is of use.—*Lancet*, 1901, vol. clx. p. 998.

Treatment of Sciatica by Means of Spinal Cocainization.—Drs. P. Marie and E. Guillani reported at the Société Médicale des Hôpitaux the case of a man, aged thirty-five years, who had suffered from an acute right-sided sciatica with well-marked and characteristic Lasègue's sign for eight days. Five-sixty-fourths of a grain of cocaine were injected beneath the spinal dura. Complete cessation of pain occurred within six minutes. The patient immediately arose and insisted on working. The pain recurred later in the day, but was less intense, and subsequently disappeared slowly. —*Gazette hebdomadaire de Medecine et de Chirurgie*, 1901, No. 27, p. 318.

Dr. R. Pulls reports the results of the intraspinal cocaine injection for the relief of a case of sciatica of a month's duration. All the well-recognized procedures were unavailing. About twenty minims of a 2 per cent. solution were injected. Many of the accompanying effects were noted. Fever and herpes labialis were also noted. The analgesia persisted for twelve hours, but the severe pains of the sciatica did not return.—*La Riforma Medica*, 1901, No. 44, p. 150.

Chemistry of Ipecac.—Drs. B. H. Paul and A. J. Cownley have been investigating the chemistry of this drug for a number of years. In their latest contribution to the study of the Brazilian variety they isolate three alkaloids—emetine, cephaeline, and psychotrine. The composition of emetine and cephaeline are comparatively well known. With reference to the new alkaloid, psychotrine, they find it in small quantities only, and it differs from these alkaloids in being sparingly soluble in ether. It is a crystalline body, melting at 280° F., and has a higher molecular weight than either emetine or cephaeline. It is readily soluble in alcohol or

chloroform, the solutions becoming dark-colored on exposure to light and depositing a dark-brown substance. With reference to the pharmacology of the earlier isolated alkaloids it is well established that both emetine and cephaeline possess powerful emetic action, the emetic dose of emetine being double, however, that of cephaeline. On the other hand, the nausea produced by cephaeline is said to be double that occasioned by emetine. Cephaeline may be used in therapy in doses of from one-tenth to one-fifth of a grain as an emetic. Emetine is better as an expectorant.—*American Journal of Pharmacy*, 1901, vol. lxxiii. pp. 57, 107.

Treatment of Endocarditis.—Dr. F. L. Newman divides the treatment of this condition in two stages—the acute and chronic. In the vast majority of the acute cases an infection must be considered against which remedial agents are for the most part powerless. If of diphtheritic origin, serum-therapy offers some hope, and if of rheumatic causation the salicylates are indicated. Complete rest of the already restless heart is a necessity. In the sthenic cases four- or five-drop doses of tincture of aconite every three or four hours and small doses of morphine, especially if there be much pain, are indicated. Cardiac weakness is, however, the dominant note, and digitalis and strychnine are desirable to quiet the irregular heart action and embarrassment. Digitalis may be left with more propriety to the later stages of the affection. Local treatment by means of the ice-bag affords great relief. In the chronic cases it is not until symptoms of dilatation appear that any medicine should be given. Up to this time the patient with compensated valvular disease should be simply enjoined to be moderate in all things—his exercise, his food, his drink, his indulgence. He must be warned particularly against all sudden violent movements, as a sudden rush for the street-car or the rapid ascent of stairs; in fact, everything that subjects the heart muscle to sudden strain. Unfortunately, in many cases of endocarditis compensation can only be partially established, and the patient is constantly threatened with cardiac failure. For such an one very much can be done by proper attention to diet, hygiene, and medication. As soon as the alarm signal is shown by disturbance of the circulation, the patient should be put to bed and kept from all exciting influences until such symptoms have disappeared. Food should be given at short intervals (four meals a day) and in small quantities, so that the stomach will be thoroughly empty before the next meal. Large quantities of fluid, particularly at meals, are to be avoided, because they increase too suddenly the amount of fluid circulating in the bloodvessels. The medicinal treatment of failure of compensation is very often quite brilliant. The objection that digitalis is contraindicated in aortic regurgitation because the lengthened diastole allows more time for the blood to run back into the ventricle, the author believes. It is easy to see that in this manner such anæmia of the brain may be produced as to cause syncope or even sudden death; but even these dangers are lost sight of as the heart becomes more enfeebled and dilated, so that the mitral valves no longer close, and the resulting venous stasis and pulmonary embarrassment demand the administration of digitalis at all hazards. In all forms of cardiac dilatation from valvular lesions, therefore, a sheet anchor is digitalis. It is absorbed slowly and eliminated more slowly, so its action

may continue for days. Strophanthus and strychnine are also indicated. Caffeine has a very uncertain, if any, action on the heart, but a very decided action on the kidneys. As a diuretic it is the most important of the heart drugs, and is, therefore, of the greatest value when there is renal suppression and dropsical effusion. Probably the most abused of the heart drugs are the nitrites. They are the most powerful reducers of blood-pressure known. They are vascular stimulants in the fullest sense. Nitroglycerin has no action on the heart, but decreases the resistance against which the systole is performed. For the palpitation the ice-bag, the belladonna plaster, and morphine are useful. The dyspnœa of heart disease has a cardinal remedy in nitroglycerin. Enlargement of the liver, beyond its treatment from the heart, can be benefited by an occasional dose of calomel or sodium phosphate, one drachm to three drachms *ter in die* in hot water. Bronchitis is largely relieved by the treatment of the heart, but occasionally codeine, morphine, or hydrocyanic acid will give relief. Anasarca should be treated promptly and the lymphatic spaces cleared. Puncturing with a large-sized needle, with all aseptic precautions, will often give the greatest relief to the patient, frequently with the disappearance of the symptoms of asthma, the œdema of the lungs, the palpitation, and other symptoms.—*Physician and Surgeon*, 1901, vol. xxiii. p. 120.

OBSTETRICS.

UNDER THE CHARGE OF

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Repeated Ectopic Gestation in the Same Patient.—LEWERS (*Transactions of the Obstetrical Society of London*, 1900, vol. xlii., Part 4) describes the case of a patient, aged twenty-nine years, who had two ectopic gestations. At the first the left tube was removed, containing a tubal mole.

The second ectopic pregnancy occurred a little more than six years later, and the right Fallopian tube and ovary and a tubal mole were removed. The ostium of the tube was widely open and the mole had been expelled into the peritoneal cavity and was lying loose at the ostium at the time of operation. The case was a complete tubal abortion.

Subcutaneous Symphysiotomy.—HERMAN (*Transactions of the Obstetrical Society of London*, 1900, vol. xlii., Part 4) reports three cases. He mentions four cases performed since this group, all of which recovered without complications. In his first patient alleged incontinence of urine persisted for some time after the operation. As examination showed no cause for this,

and as the patient when not observed was not annoyed by incontinence, it was thought to be a nervous irritability only.

The method employed was, under antiseptic precautions, to open the symphysis subcutaneously and to deliver the head by forceps. In these cases the joint was opened by a tenotomy knife, which was inserted opposite the symphysis pubis, cutting at first down and then up. Considerable separation of the pubes occurred at the moment of delivery. After the operation the parts were kept in apposition by a strong binder of webbing or muslin.

A Probable Ovarian Pregnancy.—In the *Transactions of the Obstetrical Society of London*, 1900, vol. xlii., Part 4, CROFT reports a case in which the signs and symptoms pointed to ectopic gestation. The sac and its contents were found entire and were removed by abdominal section. The patient died a few days after the operation.

On examining the specimen it contained a complete ovum and fœtus of about four months' growth, with placenta, membranes, and amniotic fluid. There was no blood or clot in the sac. The surface of the cyst contained follicles, and no separate structure corresponding to an ovary could be found upon that side. The microscopical evidence of ovarian tissue in the wall was uncertain. The Fallopian tube was present and uninjured, the fimbriated extremity was free and patent. The uterus was intact and the appendages on the opposite side were normal.

Cæsarean Section for Complete Placenta Prævia.—In the *Boston Medical and Surgical Journal*, February 14, 1901, HARE reports the case of a multipara with central placenta prævia, who was advised to go to a hospital for treatment. She declined, but sent for her physician some time later, when she was found to be having a very severe hemorrhage. The patient was finally brought to the hospital bleeding freely with a very rapid, poor pulse. The os admitted the finger-tip, and the placenta entirely covered the cervix. The child was living.

The patient was hastily prepared and at once delivered by Cæsarean section. The child weighed five and a half pounds and was resuscitated. The placenta was very large and was on the right and posterior side of the uterus. The small segment was attached to the opposite side, thus entirely covering the os. The uterus contracted well and was closed with two layers of interrupted suture of chromicized catgut. There was no bleeding from the uterus. The patient died eleven hours after the delivery of the child.

In the same journal DEWIS reports a case of central placenta prævia in a multipara, seen when very much shocked by a severe hemorrhage. The patient was delivered by version, and was freely stimulated and seemed to react. She died with symptoms of embolism.

In the same journal COGGESHALL reports a case of central placenta prævia in a multipara in labor who had been bleeding severely. The patient was given salt solution and strychnine, the cervix was rapidly dilated with considerable laceration, the placenta was separated and removed, and the child hastily delivered by version. The patient had stopped breathing, but was gradually resuscitated. The mother remained in an extremely critical condition for two days and finally recovered.

In the discussion upon these papers before the Suffolk Medical Society, the majority of opinion inclined to favor the Cæsarean operation whenever practicable.

Retroversion and Retroflexion of the Pregnant Uterus.—KEITLER reports from Chrobak's clinic in Vienna (*Monatschrift für Geburtshülfe und Gynäkologie*, 1901, Band xiii., Heft 3) a number of cases of retroversion and retroflexion of the pregnant uterus. He concludes that a backward displacement of the pregnant womb is an abnormality which may develop during pregnancy, and may not be the result of pregnancy occurring in a womb already retroverted. It is not uncommon to see the condition of retroversion become also retroflexion in these cases. They should, however, be distinguished, because both demand active treatment, and the exercise of force to secure replacement must be conducted with reference to the kind of displacement present. All cases of backward position of the uterus when the cervix is short and only the os remains are practically cases of retroversion if the mouth of the womb is directed anteriorly. Where the urine is retained the bladder must not be emptied entirely and suddenly, but very gradually. When bleeding from the bladder occurs it is evidence that necrosis of the mucous membrane has already begun.

In the *Transactions of the London Obstetrical Society*, 1900, vol. lxii., Part 4, SINCLAIR draws attention to irritability of the bladder as the symptom most constantly present in these cases.

In treatment Sinclair urges the introduction of a watch-spring pessary. The patient is made to lie upon her side with her face as far downward as possible; the uterus is restored to its place in a few hours. Sinclair has employed this method in fifteen cases in which it was universally successful. In one a fibroid tumor on the posterior wall of the uterus had fallen into Douglas' cul-de-sac and produced the symptoms of retroversion. This tumor was replaced by the pessary. Where the uterus is retroflexed and adherent, ventrofixation may be practised.

Repeated Ectopic Gestation.—In the *Annales de Gynecologie et d'Obstétrique*, March, 1901, VARNIER and SENS contribute a paper upon this subject. They have collected and examined the records of sixty-five cases, in which it seems probable that ectopic gestation occurred more than once in the same patient. They believe this to be more frequent than is usually supposed. As a rule, ectopic gestation recurs in the tube of the opposite side. In the sixty-five cases which they studied it recurred but once in the same tube. In forty-three cases the primary tubal gestation occurred sixteen times on the left side and twenty-seven times on the right. The interval of time elapsing between the second and first gestation was studied in fifty-six cases. In about one-third it varied from three to twelve months; in ten cases, from eighteen to twenty-four months; in eight cases, between two and three years, and in seven cases, between three and five years. In one case the interval was twelve years. In six cases out of the sixty-five an intra-uterine pregnancy intervened between the ectopic gestations. As regards the possibility of a third ectopic gestation in the same patient, it must be exceedingly rare, if ever observed.

An effort was made in this study to recognize conditions in the tube of the unaffected side which might predispose to ectopic gestation and to appreciate any cause other than local which might lead to this complication. In fifty-one cases forty occurred in multiparæ and eleven in nulliparæ. We should naturally expect that infection might leave in many cases alterations in the tubes predisposing to tubal gestation, but there seems to be no positive proof of this in the cases under examination. Enström describes a significant case in which the patient was treated for retroflexion, endometritis, and inflammation of the right ovary. After curettage and vaginofixation she became much better. A tubal pregnancy upon the left side followed, and at operation the right tube and ovary were found surrounded by very firm and dense adhesions. The fact that it is very difficult to determine positively by gross inspection at the time of operation that a tube is normal or diseased increases the difficulties in studying this question.

Abdominal Hysterectomy for Cancer at the Eighth Month of Pregnancy.—LEGUEU (*Comptes Rendus de la Société d'Obstétrique, de Gynecologie et de Pediatrie de Paris*, 1901, vol. iii.) reports the case of a multipara eight months pregnant with a rapidly growing epithelioma of the cervix. The vagina had not yet become involved. Free hemorrhage occurred which made it necessary to tampon the vagina and cervix. The cancer was curetted away from the cervix, to stop the hemorrhages. The mother's general condition improved, and she was submitted to operation by abdominal section. The uterus was then removed and drainage inserted, passing down through the vagina. The mother made an uninterrupted recovery. The child perished from inanition a few hours after birth.

Dystocia from Uterus Bicornis and Contracted Pelvis.—MCILWRAITH (*Canadian Practitioner*, February, 1901) reports a case of this unusual combination. The patient was a primipara who had been in labor some hours. The os admitted one finger; the cervix was not shortened. The presentation was difficult to outline, but the head was presenting and not fixed in the brim, and the back of the child was to the right. A mass of considerable size lay over the child's back, extending up to the right hypochondrium. This mass became prominent during uterine contractions, was doughy, not resonant, and the uterine souffle was heard over it very plainly. Labor proceeded in all thirty hours, with but little progress. The patient was then anæsthetized, the cervix dilated with the hand, and the occiput found posterior, but easily rotated. The internal antero-posterior diameter was found to be shortened. The patient was put in Walcher's position and an endeavor made to bring down the head by pressure. This failed, and the child was delivered by version and resuscitated. On introducing the hand to remove the placenta a septum was found extending about one-third of the distance down the interior of the uterus. The placenta had been adherent in the right cornu and the breech of the child had occupied the left. The mother made a good recovery.

Cæsarean Section for Fibrocystic Uterine Tumor.—BRODHEAD (*The Post-Graduate*, March, 1901) describes the case of a primipara having had a nor-

mal pregnancy and having a slightly contracted pelvis. On vaginal examination a tumor the size of an orange was in Douglas' cul-de-sac, pressing the cervix forward behind the pubes. An effort was made to replace the tumor with the patient in the knee-chest posture, but this failed. At full term the patient was delivered by Cæsarean section. The tumor was found to have a pedicle. It was readily ligated, and the tumor was separated from the rectum and easily removed. The cervix was dilated by the finger from above and the uterus closed. The patient made a good recovery.

Epithelioma of the Cervix Complicating Pregnancy.—In the Dublin *Journal of Medical Science*, February, 1901, KINKEAD reports the case of a multipara, eight months pregnant, having a tumor the size of a hen's egg attached to the anterior lip of the uterus by a broad pedicle. As the tumor was bleeding freely, it was removed by the curette and iron applied to the stump with a vaginal tampon. The patient recovered from the operation and was subsequently delivered of her child. She returned to the hospital, and the cancerous mass was removed from the cervix by incisions and the parts closed with silkworm-gut. A few days after the uterus was removed through the vagina. Large vessels were found at this time and the tissues were exceedingly soft. Secondary hemorrhage occurred some hours after operation, which was checked by passing a ligature around the broad ligament, tamponing the vagina, and introducing salt solution under the breasts. In several hours hemorrhage returned and required the insertion of silkworm-gut suture at the right broad ligament and the application of a clamp. The patient slowly recovered, her convalescence being complicated by an abscess in the right iliac fossa. A strip of gauze was found which had been pushed high into the pelvis. The patient ultimately made a good recovery.

GYNECOLOGY.

UNDER THE CHARGE OF

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Pelvic Massage.—OLSHAUSEN (*Centralblatt für Gynäkologie*, 1901, No. 3) concludes an extended article on this subject as follows: Massage should only be applied to the treatment of firm pelvic exudates when evidences of acute inflammation have long been absent. It is important that the masses should be readily accessible to the finger-tips. Enlarged tubes should seldom be massaged, except in rare cases in which attempts are made to empty a hydrosalpinx into the uterus.

In massaging thickened tubes the manipulation should be directed principally to the surrounding exudate.

Massage is contraindicated in the case of peritoneal adhesions, hæmatocele, and displacements of the uterus and adnexa.

Origin of Dermoid Cysts from Wolffian Bodies.—BANDLER (*Archiv für Gynäkologie*, Band lxi., No. 3) supports the theory that ovarian dermoids develop from the Wolffian bodies by reference to the fact that the epithelium of the Wolffian duct is derived from the ectoderm. He even goes so far as to affirm his belief that cystoadenomata of the ovary arise from the primordial kidneys, the tubules of which penetrate not only the hilum but the stroma of the gland. He explains the development of mixed tumors on the theory that they arise from ingrowths from the Wolffian bodies, which, on account of the prevalence of hair and skin, are recognized as dermoids.

Changes in the Endometrium due to Sclerosis of the Uterine Arteries.—SIMMONDS (*Centralblatt für Gynäkologie*, 1901, No. 3) calls attention to the frequency with which minute hemorrhages are observed in the uteri of old women. These are associated with venous thrombosis, sclerosis of the arteries, and narrowing of their lumina. The hemorrhages probably occur just before death, being due to weakened heart action, and have no clinical significance. Since these sclerotic changes are common between the ages of forty and fifty, it is fair to infer that in many cases climacteric hemorrhages are also due to this cause. Such a condition of the vessels was found by the writer in the uterus of a woman, aged fifty-four years, which was extirpated on account of obstinate bleeding, the cause of which could not be discovered.

Gynecological Operations in Diabetics.—LOMER, in a paper on this subject read before the Hamburg Obstetrical Society (*Centralblatt für Gynäkologie*, 1901, No. 3) reports a case of curettement for repeated hemorrhages in a patient whose urine at the time of the operation contained only one-tenth of 1 per cent. of sugar. Immediately after the operation 4¾ per cent. appeared, and the patient died a few days later in coma. He calls attention to the fact that in diabetics who have been weakened by loss of blood gynecological operations are especially dangerous. On the other hand, in glycosuria accompanied by obesity, amenorrhœa and pruritus the danger of coma is much less. In the discussion which followed Räther stated that he had frequently operated upon diabetics without bad results; in fact, he estimated that not less than 1 per cent. of gynecological patients had sugar in the urine. Kümmell agreed with the speaker, and believed that the unfortunate result in the case reported should be regarded as exceptional. Others emphasized the importance of testing the urine for sugar before every gynecological operation, and the dangers attending anæsthesia in diabetic patients.

Observations on Gonorrhœa in Prostitutes.—PROME (*Centralblatt für Gynäkologie*, 1901, No. 3) reports the results of the systematic hospital treatment of prostitutes in San Salvador, where the opportunities for the study of venereal diseases are apparently unequalled. Out of 407 patients 313 had gonorrhœa. The writer affirms that it is not easy to recognize gonorrhœal endometritis at its inception. Enlargement and tenderness of the uterus, with accompanying amenorrhœa, gastric disturbances, etc., point to the extension of the infection from the vagina. The adnexa were affected in 68

per cent. of all cases, 5 per cent. showing acute salpingitis; 81 per cent. were sterile.

The writer notes that nulliparæ with anteflexed uteri seem to be less prone to disease of the adnexa, and explains this on the theory that the atonic uterus in these cases does not contract at the height of the orgasm, and thus favor the draining up of gonorrhœal discharges into its cavity.

Experiments showed that infection was not produced by the contact of gonorrhœal pus with intact epithelial surfaces. Moreover, the presence of a few gonococci in the vaginal secretions of prostitutes is no proof of its infectiousness.

The writer favors curettement in acute infection of the endometrium, as a timely resort to this operation sometimes prevents extension to the tubes. In twenty subacute cases curettement caused the disappearance of exudates, and the emptying of a pyosalpinx into the uterine cavity (?), although the relief was usually only temporary. Reinfection of a patient who was discharged from the hospital as cured was rarely observed.

Sarcoma of the Cervix Uteri.—BORRMANN (*Zeitschrift für Geb. u. Gyn.*, Band xliii., Heft 2) reports the case of a patient, aged thirty years, who died of sepsis following abortion at the fourth month. At the autopsy he found a giant-celled sarcoma of the cervix, with metastases in both ovaries and the pelvic lymph glands and vessels. Although the ovaries were enlarged to the size of the fist, a corpus luteum of pregnancy was found in the centre of the tumor on the right side. He inferred that the metastases were favored by pregnancy.

Uterine Fibromyoma Complicated with Diabetes.—KLEINWACHTER (*Zeitschrift für Geb. u. Gyn.*, Band xliii., Heft 2) reports two cases, four only having previously been recorded in the literature. In his cases, unlike the others, the tumor was small, the hemorrhages slight, and the percentage of sugar was low.

Thrombosis and Embolism Following Hysteromyomectomy.—BURCKHARDT (*Zeitschrift für Geb. u. Gyn.*, Band xliii., Heft 2) met with twelve cases (six fatal) in the course of 236 operations. Œdema of one or both of the lower limbs was noted as the principal indication of thrombosis in one-half of the cases. Mahler's sign was present in eight; on the other hand, this phenomenon was noted in cases which presented no other evidences of venous thrombosis.

Diagnosis of Cancer of the Body of the Uterus.—HANDFIELD-JONES (*British Medical Journal*, January 19, 1901) summarizes as follows: 1. Cancer of the body of the uterus is preceded by a stage of benign adenoma. 2. The microscopical examination of tissue obtained by the curette is apt to be misleading in the early stage of the disease, since the superficial epithelium alone is removed and not the glands. 3. Hence, clinical evidence is more useful than microscopical. 4. There is a wide variation in the degree of malignancy. 5. Rapid enlargement of the uterus is the most positive indication for hysterectomy.

OTOLOGY.

UNDER THE CHARGE OF
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Persistent Retroauricular Opening after Radical Operations and Subsequent Plastic Closure.—F. TRAUTMANN (*Archiv f. Ohrenh.*, vol. xlvii.) favors persistent retroauricular opening after the radical operation as the best means of carrying on the treatment necessary to bring about epidermization of the new formed bone cavity. Treatment for this purpose carried on by means of tampons of sterilized gauze, through the auditory meatus alone, is very painful to the patient and unsatisfactory to the surgeon. In performing a radical operation on the mastoid antrum and middle ear cavities, involving removal of the posterior wall of the auditory canal, Trautmann makes the first retroauricular incision ¾ cm. behind the insertion of the auricle. The soft tissues with the periosteum of the mastoid are pushed back and the surface of the bone fully exposed, and the cutaneous auditory canal, along its upper, posterior, and inferior wall is removed by a raspatory from the bony canal. If the remnant of the membrana tympani is large its excision must be performed with a small knife. Further operation on the mastoid and posterior wall of the bony canal must depend upon the apparent position of the sinus. If the latter lies far forward, as it does in a narrow, high mastoid, the bone must be opened first at the attic and aditus, the malleus and incus, and granulations removed, and the surgeon must then pass backward toward the antrum by careful removal of thin lamellæ of bone. If the mastoid is flat and wide, the sinus lies far behind, and then the antrum may be opened at once, and the surgeon pass forward into the attic, by removal of lamellæ of bone, and remove the malleus, incus, and granulations. After the new bone cavity has been fully made the cutaneous auditory canal is transplanted. This is important as regards the persistent retroauricular wound. The splitting of the posterior wall of the cutaneous canal and the formation thus of the so-called cutaneous flaps for lining the inner and posterior wall of the new formed bony cavity are best accomplished, according to Trautmann, by inserting a species of director, made by having a groove in the handle of a cotton holder thick enough to nearly fill the cutaneous canal. This groove in the director is placed so as to run on the upper posterior wall of the canal. Here the incision is made so as to form an upper narrow and a lower broad flap. The incision is carried 5 mm. into the skin of the concha, the skin pushed away from the cartilage, and at the end of the canal incision, an incision made at right angles to it, close to but not through the cartilage of the concha. Thus two rectangular skin flaps are formed. The lower one is stitched into the lower angle of the mastoid wound, but not at its most external limit, and the upper flap, the narrow one, is tamponed into position in the upper part of the bone cavity. Then the upper and lower angles of the wound are brought together with one or

two sutures. The edges of the auricular wound are allowed to heal by granulations in Trautmann's operation. By this means the retroauricular wound is rendered somewhat smaller by reason of shrinkage and epidermization. The incisions carried into the concha in this method enable the surgeon by tamponading to widen the meatus somewhat without deforming it, and to carry on some of the treatment with tampons through the widened meatus. The tampons consist of acetate of aluminium gauze. These are allowed to remain as long as possible in position. If the dressings become moist in spite of the acetate of aluminium gauze, then 10 per cent. xeroform (tribromo-carbolate of bismuth) gauze may be tried. Xeroform gauze gets black by the decomposing effects of the pus, but as the suppuration diminishes the discoloration of the xeroform lessens, and serves as an indication of the decreasing suppuration. Iodoform gauze is not used by Trautmann, because it irritates the skin, especially in children. If the insertion of the tampons is painful the wound may be dusted with orthoform, which seems to lessen the pain in most cases. Epidermization proceeds slowly in chronic cases, in which the mucous membrane is infiltrated and hyperæmic and chronic nasal catarrh is present. Constitutional taints exercise a disadvantageous influence on healing and must receive a general medication. Epidermization is aided by skin grafting after Thiersch's method. In some cases secretion occurs in the finally epidermized cavity, in connection with acute nasal catarrh. Such processes can be healed by simple dry treatment. Those cases epidermize most quickly in which the caries is limited to the hammer and anvil, with nothing more than granulations in the attic, aditus, and antrum, and without complications and constitutional disorder. Also cases of cholesteatoma without complication quickly epidermize. After complete healing superficial suppuration may occur in the new epithelium. This can be prevented by applying to the bony cavity once a week a thin layer of ointment of the white precipitate of mercury in sterilized vaseline (2 per cent.), and then packing this cavity loosely with sterile gauze. If the tympanic mucous membrane is not swollen at the outset, healing occurs with conservation of all the delicate contours of the inner wall of the drum cavity, excepting the stapes. This is rarely seen after healing of the wound of the radical operation, as its niche is covered with new epidermis that greatly interferes with its mobility and visibility.

If the mucous membrane is swollen before the operation it remains after the operation, thickened by increase in connective tissue. There exists, however, a narrow opening in this new thick tympanic wall communicating with the Eustachian tube. Through this there may be an escape of mucus upon the occurrence of an acute nasal catarrh.

Trautmann states (*loc. cit.*, p. 6) that galvano-cauterization of the edges of this opening followed by firm tamponade against the labyrinth wall or inner wall of the drum cavity will effect its closure. He also says that galvano-cauterization of the pharyngeal mouth of the Eustachian tube will not prevent communication between the middle ear and the nasopharynx. Sometimes there is found a new membrane having the appearance of a new membrana tympani. This is not formed from the remnant of the old membrane, however, but is a neoplastic connective tissue membrane.

In the region of the aditus and attic repullulating ganulations easily form

and prevent epidermization. If these granulations are allowed to remain in position without proper treatment they may become organized and covered with epidermis so that healing has apparently occurred; but beneath such granulations suppuration will continue. Therefore, these granulations must be removed and their occurrence prevented. Their presence by incarcerating the stapes interferes greatly with any hearing that may remain. Tamponade widens the auditory canal and new formed bony cavity, but the canal narrows again after the tamponade is left out. Narrowing also takes place in the retroauricular wound, in some cases almost to closure of the same. The average depth of the new-formed cavity varies in children up to fourteen years of age from 2½ cm. to 3 cm.; in adults from 3½ cm. to 3¾ cm. The average diameters, vertical and transverse, of the retroauricular opening, both in children and adults, are about the same, and amount to from 1 cm. to 1.5 cm. Whether and when the retroauricular opening is closed by a plastic operation depends upon, first, the patient, and, secondly, upon the nature of the malady inducing the operation, and, thirdly, upon the healing process.

The long continued treatment, necessary to induce healing of the middle ear cavities, renders most patients unwilling to undergo even a short second operation for plastic closure of the wound behind the auricle. This wound should not be closed until definite healing has taken place. If cholesteatoma was the reason for the operation the retroauricular opening should not be closed. If it is decided to close the opening behind the auricle it is advisable not to do this until at least a year has elapsed from the time of the original radical operation.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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Foreign Bodies in the Larynx.—Dr. A. W. de Roaldes reports (*New York Medical Journal*, October 6, 1900) a case of a pin in the larynx of a child nine years of age which had been swallowed two years previously and which he removed by endolaryngeal procedures.

Thyroid Tissue in the Larynx and Trachea.—Dr. Otto T. Freer reports (*Journal of the American Medical Association*, March 30, 1901) a case of this rare condition, stated to be the tenth on record. The patient was a woman, aged thirty-two years, who had had attacks of hoarseness every second or third winter from childhood. After a hoarseness of several weeks suffocation and wheezing paroxysms occurred for the first time and recurred during subsequent attacks. The larynx was found normal down to the true vocal bands, but just below them was a protruding ledge of tissue forming

an irregular ring with much the contour of a second pair of vocal bands below the true ones. The thickening extended along the trachea for three or four rings, having a prominent tumor projecting from the posterior wall. Palpation showed almost complete absence of the thyroid gland. An attempt at destruction of the tissue by electrolysis was followed on the second day with tumefaction to such an extent as to render immediate tracheotomy necessary. A portion of the growth on the posterior wall of the trachea was subsequently removed with the wire snare and forceps, and when examined under the microscope proved to be typical thyroid tissue. The patient refused operative measures by access from without, and was content to live with her tracheotomy tube in position.

Angioma of the Larynx.—In the course of an article upon this subject, Prof. SEIFERT, of Wurzburg, reports (*Revue hebdomadaire de Laryngologie, d' Otologie et de Rhinologie*, January 12, 1901) a case of this rare neoplasm in a man, aged fifty years. The growth occupied the anterior commissure of the glottis, its surface of insertion extending as far as the subglottic region, rendering removal of the tumor, which was accomplished with forceps, rather difficult. Hemorrhage was considerable, but became arrested at the end of ten minutes after the patient had swallowed a great deal of ice. The voice immediately became clear and recovery was uninterrupted.

MR. A. J. BRADY reports (*Journal of Laryngology, Rhinology, and Otology*, January, 1901) an angioma the size of a cherry below the anterior commissure of the vocal cords of a lad, aged six years. Several attempts at operating under cocaine were made without avail, even after a whole month's training. Under chloroform, and drawing the tongue forward with a Kirsten tongue depressor so as to expose a view of the upper portion of the glottis, though not of the growth, the latter was successfully removed with a Heryng's laryngeal curette with but slight hemorrhage, some remnants being removed in a similar way ten days later.

Emphysema of the Upper Eyelid from Nasal Lesions.—PROF. BEAMAN DOUGLASS, of New York, discusses this subject (*New York Medical Journal*, March 23, 1901) and reports four cases of his own, two of which occurred in immediate sequence to operation upon the ethmoid bone and two independently of operation.

The emphysema of the upper eyelid is regarded as probably due to perforation of the wall of the orbit near the middle, whence the air at once enters the space between the orbital periosteum and the first fascia of the eyeball. The subject is discussed by the writer in several aspects, especially the medico-legal one, concerning which he concludes that the surgeon is not at fault for emphysema following operation. Nevertheless, he comes to two important conclusions: First, that the curette should be avoided as much as possible, for he believes that it is the curette that is responsible for much of the traumatism occurring to the lamina papyracea. Much better work can be done with the alligator forceps or some kind of cutting forceps which is less apt to perforate, and which will, with equal success, drain pus or remove polyps as well as the mucous membrane lining the ethmoid cells.

His second rule is never to amputate any part of the middle turbinate, as

it forms a very important guide along which an operation may be carried out without risking entrance either in the orbit or brain cavity in operations on the ethmoid. Finally, the statement is made that abscess, destruction of the eye, and possible meningitis may arise from septic conditions in operations carelessly done upon the ethmoid bone.

[Dr. Douglass' warning is quite justifiable, for the compiler is aware of fatal meningitis following what was described as merely exploratory procedures in the region of the ethmoid cells.]

A New Method of Extirpating Growths from the Larynx of Children.—Dr. Mouselles, of Florence, extols (*La Semaine Méd.*, December, 1900; *Revue hebdomadaire de Laryngologie, d'Otologie et de Rhinologie*, March 16, 1901) a device of his own which consists in inserting an O'Dwyer intubation tube, fenestrated toward its lower extremity, and scraping the larynx through the aperture. The tube can be turned in any direction while remaining fixed to the introducer.

Removal of a Foreign Body Through the Bronchus by Intrathoracic Tracheotomy.—Mr. H. Milton (*The Lancet*, January 29, 1901) performed this operation upon a man, aged forty years, for removal of a tubular portion of a tracheal canula which he had been wearing for some years on account of syphilitic stenosis of the larynx. The sternum was exposed, sawed through vertically in the middle line, and the fragments separated by powerful retractors. The trachea was pulled upward with a hook so as to expose the bifurcation, above which an incision was made. The little finger when inserted into the right bronchus impinged upon the foreign body, which was eventually removed with forceps guided along the finger. Unfortunately, the case terminated fatally, in consequence of pre-existing sepsis.

The method of performing this operation and the after-treatment are discussed in detail.

Post-influenzal Tracheal Hemorrhage.—Mr. James Donelan reports (*Journal of Laryngology, Rhinology, and Otology*, January, 1901) five cases, and alludes to four others, the diagnosis resting principally upon the detection, under laryngoscopic inspection, of more or less blood upon the wall of the trachea.

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